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1875. Hime, T. W., M.D., 54, Horton Road, Bradford, Yorkshire.
1875. Home, Sir Anthony D., K.C.B., V.C., M.D., Surg.-Gen. (*retired*) 9, Denmark Terrace, Brighton.
1887. Hunt, J. P., Surg.-Major, care of The Admiralty, Whitehall, S.W.
1881. Irwin, Chamney Graves, M.D. (*retired*), Dep.-Surg.-Gen., Casa Hatfield, St. Albans, Herts.
1888. Karkeek, P. Q., Isca, Torquay, Devon.
1887. Kelly, C., M.D., Gratwicke Road, Worthing.
1883. Kirker, Gilbert, R.N., Staff-Surg., M.D., R. N. Hosp., Gosport.
1886. Lingard, A., M.B., Imperial Bacteriologist, Muktesar, Kumâon Hills, N.W.P., India.
1885. Lloyd, W. Harris, M.D., Inspec.-Gen. R.N. (*gone abroad*).
1890. Luard, H. B., Surg.-Capt., Purfleet Rectory.
1881. Lyons, R. T., M.D., Brig.-Surg. (*retired*), Cherry Hinton Hall, near Cambridge.
1860. May, George, M.B., F.R.C.S., 43, Castle Street, Reading, Berks.
1890. M'Neill, R., M.D., Argyll County Council, Oban, N.B.
1881. Oldham, Chas., Brigade-Surg. Ind. Med. Dept. (*retired*), The Lodge, Great Bealings, Woodbridge, Suffolk.
1891. Osborne, Frank, 7, Archway Road, Highgate.
1874. Reid, Walter, M.D., Dep. Inspector-General, Royal Naval Hospital, Haslar.
1863. Rowe, T. Smith, M.D., 1, Union Crescent, Margate.
1881. Russell, E. G., M.B., B.Sc., Surg. Lieut.-Col., I.M.S. Bengal Army.
1859. Sanderson, J. Burdon, M.D., F.R.S., 64, Banbury Road, Oxford.
1884. Simpson, W. J., M.D., 14, Gloucester Place, Portman Sq., W.
1854. Spinks, C. N., 3, Palmyra Square, Warrington.
1889. Street, A. F., M.D., St. Mildred's Road, Westgate-on-Sea, Kent.

Elected
1885. Sturridge, P. F., Stramongate, Kendal, Westmorland.
1885. Tomes, Arthur, Surg. Lieut.-Col. Ind. Med. Service, Bengal.
1876. Turner, G., M.D., Cape Town, South Africa.
1879. Watson, Alexander, M.D., R.N., Insp.-Gen., Army and Navy Club.
1893. Whiston, P. H., Surg.-Capt., The Palace, Rochester, Kent.
1854. Wiblin, John, Northleigh, Wimborne, Dorset.

Corresponding Members.

1875. Arkhangelsky, D., M.D., St. Petersburg, Russia.
1853. Beneke, F. W., Marburg, Germany.
1860. Brionde, A. de (*address unknown*).
1894. Catrin, L., Dr., Val-de-Grâce, Paris.
1856. Cox, Wm. Isidore, Hawkesbury-Upton, Chippenham, Gloucestershire.
1871. Cuningham, J. M., M.D., C.S.I., Surg.-Gen. Ind. Med. Dept., Sanitary Commissioner of Govt. of India.
1894. Davidson, Dr., Kevock, 30, Morningside Drive, Edinburgh.
1878. Delmas, Louis H., M.D., P.O. Box 524, Havana, Cuba.
1867. Dickson, E. Dalziel, M.D., Physician to British Embassy, Constantinople.
1895. Gache, Samuel, M.D., Buenos Ayres, Argentine Republic.
1892. Hauser, Philip, M.D., Madrid.
1889. Hewitt, Chas. N., M.D., Red Wing, Minnesota, U.S.A.
1886. Janssens, E., M.D., Inspecteur en Chef d'Hygiène, 1, Rue de Lombard 21, Brussels.
1881. Kynsey, W. R., C.M.G., Colombo, Ceylon.
1857. Leas, Jose Fernandes da Silva, Cape de Verde.
1875. Lent, —, M.D., Cologne.
1886. Le Roi de Méricourt, L., M.D., 5, Rue Cambacères, Paris.
1870. Mallet, —, M.D., Warsaw.
1851. Monzon, Rafael, Barbacaos, New Grenada.
1894. Netter, Dr. Arnould, 129, Boulevard St. Germain, Paris.
1854. Purple, S. S., M.D., New York, U.S.A.
1886. Rochard, G., M.D., Paris.
1879. Sarell, Richard, M.D., Pera and Therapia, Constantinople.
1873. Schleisner, —, M.D., Copenhagen.
1874. Schlimmel, —, M.D., Teheran.
1888. Thompson, Ashburton, M.D., Sydney, New South Wales.
1851. Tribes, E., Nismes, France.
1853. Valerj, —, M.D., Rome.
1855. Virchow, Rudolf, Professor, 10, Schellings Strasse, Berlin.
1856. Von Iffland, —, Beaufort, New Quebec.
1861. Whitelaw, Wm., M.D., Kirkintilloch, Dumbartonshire.

Jenner Medallist.

1898. William Henry Power, F.R.S.

TRANSACTIONS

OF

The Epidemiological Society of London.

INAUGURAL ADDRESS OF SESSION 1898–99.

Half a Century of Sanitary Progress, and its Results.

By H. FRANKLIN PARSONS, M.D., President.

(*Read: November 18th*, 1898).

Gentlemen,—In the first place let me thank you for the honour which you have done me in electing me President of the Epidemiological Society: an honour which I should scarcely have felt myself entitled to accept, had I not regarded it as intended for the Medical Department of the Local Government Board rather than for myself personally in virtue of any merits or qualifications of my own for the position. Conscious as I am of the difficulty of following worthily in the footsteps of the distinguished men who have preceded me in the office of President, I must crave your kind indulgence, and ask you to take the will for the deed.

In casting about for a subject on which I might profitably address you this evening, I remembered that it is now fifty years since the first general Public Health Act, that of 1848, was passed. The Annual Report of the Registrar General for 1896, published this year, completes also a fifty years' record of the deaths from different causes in England and Wales; since the classification of the causes of death, which had been suspended for several years, was resumed in 1847. It has occurred to me, therefore, that in this age of Jubilee celebrations it might not be inappropriate to take stock of our position, and endeavour to ascertain what measure of success has attended the legislative and administrative efforts which have been made during the past half century to improve the sanitary condition of this country, and to ward off the causes of prevent able—and especially of epidemic—diseases. In so doing I am well aware that a similar task has been already

performed, much better than I can do it, by my distinguished official chief and predecessor in this chair, Sir Richard Thorne, in his Presidential Address in 1887, on the Progress of Preventive Medicine in the Victorian era. I hope, however, to avoid taking you over quite the same ground.

Of the sanitary condition of England and Wales some half century ago, and more especially of the conditions of life in the poorer quarters of the larger towns, graphic accounts are to be found in the Reports of the Health of Towns Commission, published in 1844-5, which reveal a state of things almost incredible to us at the present day. Of fifty towns examined by the Commission, in scarcely one could the sewerage or drainage be considered complete and good, even according to the standard of that day; while in seven it was indifferent, and in forty-two decidedly bad as regards the districts inhabited by the poorer classes. In cases where the drainage of a town had been locally described as good, it was often found that only the principal streets had sewers, and even in those streets the houses were but imperfectly provided with house drains; while the most crowded portions of the town, those most densely inhabited by the poorer classes, were utterly neglected and had no drainage: the refuse being allowed to accumulate and decompose in open channels and pools, or to run into open and stagnant ditches in the immediate vicinity of the houses. Even where drainage works had been executed, the greater part had been constructed only on demands for the removal of pressing inconveniences and for the drainage of particular places. Moreover, the sewers were for the reception of surface water only: in some of the larger and most crowded towns all entrance into the sewers of house drains or drains from water-closets was prohibited under penalty; in others the entrance of house drains into the sewer was deemed the concession of a privilege, and was subject to regulations and payments which confined the advantage to the wealthy. Such sewers and drains as there were, were of imperfect construction, of brick or stone, commonly with flat bottoms—the use of earthenware pipes being mentioned as a novelty—and the inlets were untrapped and a source of noisome stenches. The form of privy in vogue at that time is not explicitly described, but it involved the storage of excremental filth in large quantities and for prolonged periods in the neighbourhood of houses—often, indeed, immediately under them. The houses of the wealthier classes were furnished with water-

closets discharging into cesspools, while elsewhere privies were in general use; these were often badly placed, and shared by a large number of houses. As regards water supply, in the great majority of the towns investigated, the mains were carried only through the principal streets. In a large proportion of the poorer districts the inhabitants had only out-door supplies by means of stand-pipes, or common tanks or wells; being in many instances obliged to fetch water from considerable distances, at much inconvenience, delay, labour and expense. In many towns, indeed, they were dependent for supplies either on collections of rain-water, or on water taken from adjacent streams, or pumped from springs frequently liable to be polluted Where a water-service existed the supply was commonly an intermittent one, which rendered necessary the use of water-butts or tanks, in which the water was liable to be deteriorated by impurities. Thus, a condition of things similar to that which during the recent scarcity of water in East London has given rise to such loud and general complaints, was fifty years ago quite the usual one.

One result of this universal fouling of the air, the soil, and the water supply, was the constant prevalence of fever. Dr. Southwood Smith in his evidence, referring especially to East London, says: "In every district in which fever returns frequently, and prevails extensively, there is uniformly bad sewerage and bad supply of water, a bad supply of scavengers, and a consequent accumulation of filth. The streets, courts, alleys, and houses in which fever first breaks out, and in which it becomes most prevalent and fatal, are invariably those in the immediate neighbourhood of uncovered sewers, stagnant ditches and ponds, gutters always full of putrefying sewage, nightmen's yards and privies, the soil of which lies openly exposed, and is seldom or never removed. It is not possible for any language to convey an adequate conception of the poisonous condition in which large portions of these districts always remain, winter and summer, in dry and rainy seasons, from the masses of putrefying matter which are allowed to accumulate."

Other medical evidence before the Commission showed that defective drainage, neglect of house and street cleaning, bad ventilation and imperfect supplies of water, contributed to produce atmospheric impurities which affected the general health and physical condition of the population: generating acute, chronic, and ultimately organic disease, especially scrofulous affections and consumption, in addi-

tion to the fevers and other forms of disease to which public attention had hitherto been chiefly directed.

At the time when this enquiry was made the distinction between typhus and enteric fevers had not been established, nor the propagation of the latter by the medium of specifically polluted drinking water ascertained; hence polluted water supply as a cause of disease receives less notice in these reports than insufficiency of supply for purposes of domestic cleanliness and drain flushing. There is reason to think that the prevalent fever comprised both typhus and enteric fever. The association of prevalence of phthisis and scrofula with filth conditions is of interest, in relation to modern views concerning the propagation of tuberculosis.

Among other unwholesome conditions then prevalent in the poorer parts of towns, upon which stress is laid by the Commissioners as conducing to disease and death, may be mentioned the want of surface cleanliness, especially in courts; nuisances from slaughter-houses, pigstyes and dunghills in crowded localities; crowding of houses on the ground without sufficient space for the access of light and circulation of air; cellar dwellings; overcrowding; and want of proper ventilation in dwelling-houses, schools and factories.

Nor were these evils confined to the towns, for, as the Commissioners say, "The most important evils affecting the public health throughout England and Wales are characterised by little variety, and it is only in the degree of their intensity that the towns exhibit the worst examples of such evils. Villages and clusters of houses inhabited by the poor are often under the influence of the same causes of disease, though their effect in such situations may be frequently rendered comparatively slight from the more free circulation of the external air. The vitiation of the atmosphere, from overcrowding and the absence of proper ventilation in individual apartments, produces in the rural districts the same disease that arises from the same causes in a town population."

In England and Wales, during the five years 1838-42, the average death-rate per 1,000 inhabitants was 22.1, of which 1.053, or 4 per cent., was due to "fever"; and in the four years 1847-50, out of an average annual death-rate of 23.4 per 1,000 inhabitants, 1.246, or 5.3 per cent. was due to fever. On the other hand, in the five years ending 1896, of an average annual death-rate reduced to 18.1 per 1,000 inhabitants, little more than 1 per cent. (.184) has been caused by the several forms of continued fever (see

Chart II). In London the death-rate from "fever" per 1,000 inhabitants was 1.339 in the three years 1838-40, and .979 in the ten years 1841-50; whereas in the five years ending 1896 it had fallen to .142.

Yet, grossly unwholesome as were the conditions under which the poorer inhabitants of our towns lived half a century ago, and great as was the consequent waste of human life, the health of the kingdom was even then far better than it had been in earlier times. Lord Macaulay, in his well-known comparison between the state of England at the death of Charles II in 1685, and that at the time when his History was written, in the middle of the present century, says: "Some frightful diseases have now been extirpated by science, and some have been banished by police. The term of human life has been lengthened over the whole kingdom, and especially in the towns. The year 1685 was not accounted sickly; yet in the year 1685 more than 1 in 23 of the inhabitants of the capital died. At present only one inhabitant of the capital in forty dies annually. The difference in salubrity between the London of the nineteenth century and the London of the seventeenth century is very far greater than the difference between London in an ordinary year and London in a year of cholera."

It is to be observed, lest we should be inclined to overestimate the effect of sanitary legislation, that the improved health of the country to which Lord Macaulay refers was due to the improved social conditions brought about by the increase of knowledge and the growth of civilisation, rather than to legislative or administrative efforts consciously directed towards the promotion of the public health. Before, therefore, we consider the action which has been taken during the past fifty years to improve the sanitary state of the country, and its effect upon the mortality and prevalence of epidemic diseases, it will be as well to glance at the social changes which have been in progress during the same period.

It will hardly be denied that the general prosperity and well-being of the masses of the population of this country has on the whole advanced during the past half century. Mr. Henry George, indeed, has said that under our present social system the rich are growing richer, and the poor are growing poorer. Whether or not this may be true of the extremes of the social scale, we need not stop to enquire; but it seems unquestionable that the intermediate classes, those in that happy position removed from the extremes of poverty and riches, in which the prophet prayed that

his lot might be cast, and more especially the great wage-earning class, are on the whole better off than they were fifty years ago. Wages have risen, the hours of labour have been shortened, and the necessaries of life have become cheaper, either actually or relatively to the wages earned. If house-rent has not fallen, the houses built for the working classes are healthier and more convenient than those built fifty years ago. The average price of wheat per quarter, which was 46*s.* 8*d.* in the five years 1847-51, and 62*s.* 1*d.* during the five years 1852-6 (which included those of the Russian war), was only 25*s.* 9*d.* in the five years 1892-6. The introduction of preserved foods from the colonies and abroad has increased the food supply, and brought a larger variety within general reach. Vegetables, fruit, and milk are more plentiful in our towns than formerly, and meat is in more general use. Unfortunately, the cheapening of agricultural produce has its darker side in the depression from which the agricultural classes have suffered during the past twenty years. Some other classes have also suffered through similar economic causes, *e.g.*, tin-, copper-, and lead-miners, and sugar refiners. But, owing to the increased means of communication, the effect of such depression in particular callings has been to cause the transference of labour to other callings, or to promote emigration, rather than to bring about anything approaching famine. There have been temporary and local periods of scarcity, due to loss of employment through strikes and severe seasons; and there is of course always more or less chronic distress in the poor quarters of our large towns; but since the cotton famine in Lancashire in the early sixties, there has been, so far as I am aware, no period of extensive scarcity in this country sufficient to produce a marked effect upon the public health. The proportion of persons receiving poor law relief per 1,000 population has fallen from 57 in the three years 1849-51 to 23.4 in the five years 1892-6. Prosperity, of course, has not been uninterrupted; there was a period of great prosperity in the later sixties and early seventies, followed by a period of depression in the late seventies and early eighties. The marriage rate and the birth rate have somewhat declined in recent years. (See Chart I.) Marriages of persons under twenty-one years of age became more frequent during the first half of the period, and were most numerous during the seventies; since then they have fallen off, but are still more frequent than in the late forties. The proportion of children born out of

wedlock has declined from 6.7 per cent. in the quinquennium 1846-50 to 4.2 per cent. in 1891-95. With the progress of education, the proportion of persons who signed the marriage register by mark has continuously declined—that of men from 312 per 1,000 in 1847 to 37 in 1896; and that of women from 455 in 1847 and 462 in 1850 to 43 in 1896. Education has become more general, especially since the passing of the Education Act of 1870, and its spread has done much to diminish the ignorance and prejudice which too often impeded the earlier efforts for the promotion of the public health. We may also note an increased sense of the responsibility of society for the welfare of its poorer and more helpless members.

Nor among the causes tending to the reduction of mortality must mention be omitted of the progress of curative medicine and surgery. For instance, the introduction of the antiseptic system, which has diminished so greatly the danger of surgical operations, can hardly have failed to produce an appreciable effect upon the death-rate. Deaths from accident and negligence have fallen from an average rate of 689 per million in the five years 1861-65 to one of 563.6 in the five years 1891-95, notwithstanding the increased use of machinery and the growing congestion of traffic in our streets. During the same period, the death-rate per million from diseases of organs of locomotion has fallen from 106.4 to 62.4, and that from diseases of the organs of generation from 62.0 to 48.6; both of these classes comprising diseases specially amenable to surgical treatment.

But side by side with the social changes favourable to the public health which I have mentioned, there has been one marked change tending in the contrary direction. I refer to the increasing aggregation of the population in the large towns, and the depopulation of the agricultural districts.

At the census of 1851 the population of England and Wales was nearly equally divided between the towns and country districts, 8,990,809 persons residing in the towns and 8,936,800 residing in country places. At the census of 1891, 20,895,504 persons resided in urban sanitary districts, and only 8,107,021 in rural sanitary districts. The proportion of persons living in urban districts to those living in rural districts in 1891 was 258 to 100; having risen from one of 212 in urban to 100 in rural districts at the census of 1881. The relative proportion of urban to rural population will, of course, vary according to the standard taken of what

constitutes a town. Many of the smaller "urban" districts are in fact mere villages, indistinguishable in character from those in the surrounding 'rural" districts, and if these be included among the rural population, the proportion of the latter will be increased; but on the other hand there are so-called "rural" districts with large and closely aggregated manufacturing and mining populations, living under urban conditions, though without the advantages of urban administration. The aggregate population of the rural sanitary districts in England and Wales increased by about 3 per cent. between 1881 and 1891; but in many of the counties the population of the rural districts declined during that period, though the falling-off in these was counterbalanced by the increase in population of rural districts in other counties, more especially in the manufacturing and mining parts of the country, and in the "residential" counties in the neighbourhood of London and other large towns.

At the census of 1891 the population of London exceeded the number of persons resident in England and Wales who were returned as born in London by 18 per cent., and a similar excess occurred in the manufacturing and mining counties; in Yorkshire it amounted to 6·5 per cent., in Lancashire to 19 per cent., and in Glamorganshire to 42 per cent, of the natives. On the other hand, in the case of the agricultural counties, the number of persons resident in England and Wales returned as born in those counties exceeded the resident population: in Huntingdonshire by as much as 38 per cent. of the latter. Again, the number of persons in England and Wales returned as agricultural labourers diminished from 870,798 in 1881 to 780,707 in 1891, a falling-off of 10.3 per cent. The occupation of an agricultural labourer, in spite of its hardships, is a very healthy one; its comparative mortality figure is only 66; that of all males being 100. In other words, only two agricultural labourers die, where in the general population among equal numbers of corresponding ages three males would die. It is evident, therefore, that the withdrawal from the population of so large a healthy constituent, or the transference of its members to other and less healthful fields of labour, must unfavourably affect the general death-rate of the country.

Among the causes which have brought about this transference of population from country to town may be mentioned the agricultural depression, the supersession of manual labour by machinery, and of water-power by steam,

and the growing preference for the advantages and excitements of a town life over the more monotonous if healthier country life. I do not think that it can be justly ascribed to our modern works of town drainage and water supply. The experience of other times and other lands shows that where the need of defence, commercial and industrial convenience, or social advantages, tends to collect people into towns, considerations of health are powerless to prevent this aggregation. Queen Elizabeth thought that London was too big in her day, and passed a law that no more houses should be built; so also did Oliver Cromwell: yet neither of these powerful rulers was able to restrain its increase. It is not the sanitary engineer who induces people to crowd together in towns. All that he does is to enable more people to live, in a tolerable state of health, on an acre or a square mile of ground, than could have existed there at all if all their refuse had had to be deposited on the area, and their water supply obtained from it.

But, nevertheless, the environments of people crowded together in towns—

> "In populous city pent,
> Where houses thick and sewers annoy the air,"

are, *cæteris paribus*, far less wholesome than those of persons living in the open country; the impurities resulting from the effete products of human beings are concentrated in a smaller compass; the purifying powers of vegetation and of the virgin earth are more or less wanting, owing to the ground being covered with buildings, roadways, and pavements; the wholesome rays of the sun are obstructed by smoke and dust; the circulation of the air is impeded; and the facilities for the propagation of infectious diseases are increased by the more frequent and closer contact of each individual with others. Hence the death-rate of urban districts tends to be constantly higher than that of rural districts. In England and Wales, during the ten years ending 1896, the average annual death-rate per 1000 estimated population was in town districts 19.5, and in country districts 16.9; while in earlier periods the difference in favour of the country was greater still. Moreover, a comparison of the crude death-rates of town and country districts does not exhibit to the full the greater salubrity of the latter; for it is commonly the younger adults, vigorous in body and mind, and in the actuarial sense, "better lives," who migrate to towns, leaving the older and less active and enterprising members in the country districts. Hence the age constitution of a rural population, as compared with that of an

urban population, is commonly such that, other things being equal, the former should experience the higher death-rate and the latter the lower; whereas, in actual fact, the reverse is the case; and when the death-rates for urban and rural districts are corrected for the age and sex constitution of the respective populations, the difference in favour of the latter is increased. Thus, to quote from a table in the Supplement to the Fifty-fifth Annual Report of the Registrar-General, the mean annual death-rate in England and Wales during the ten years 1881-90 was 19.08. In "urban England," viz., the aggregate of the seventy-eight principal towns, the death-rate was 21.08, and in rural England, comprising the remainder of the kingdom, it was 17.63. When, however, these crude death-rates are corrected for age and sex distribution, the urban rate is increased to 22.32, and the rural rate reduced to 16.95. In individual counties the effect of correction for age and sex differences is still greater: thus, for Huntingdonshire the comparative mortality figure is 730, and in Lancashire 1,268, that of England and Wales being 1,000: *i.e.*, to reduce the crude death-rates in these counties to a standard population, the death-rate in the former would have to be diminished by more than a fourth and that in the latter increased by more than a fourth.

The relation between density of population and mortality is well illustrated by a table in the same Supplement, in which groups of registration districts are arranged in order of their death-rates, corrected to a standard population. In the group of districts having the lowest corrected death-rate, viz., between 12 and 13 per 1,000 population, the density was 138 persons to a square mile; whereas in the group of districts having a corrected death-rate of 30 to 36 per 1,000, the density of population was 19,584 persons per square mile; and between these two extremes the corrected death-rate increased with the density in almost uninterrupted gradation.

As the density of the estimated population in England and Wales increased from 325 persons per square mile in 1851-60 to 470 in 1881-90 and 527 in 1896, and as the increase was not spread uniformly over the country but took place mainly in the towns, it is clear that the tendency of the change in the distribution of the population, apart from sanitary improvements, would have been to increase the death-rate and also the deaths from the different forms of infectious diseases and the infant mortality, all of which, in the Supplement from which I have quoted, are

shown to occasion a higher mortality in urban than in rural England.

The spread of infectious diseases is also favoured by the collection of children in schools for the purpose of elementary education: a condition which has greatly increased since the passing of the Education Act of 1870.

It may be feared, too, that modern ethical tendencies are not wholly in a direction to enhance a sense of parental duty, or of the value of infant life.

Let us now consider the efforts which have been made by the legislature during the course of the past fifty years for the protection of the public health. The era of sanitary legislation in this country may be said to have been inaugurated by the Public Health Act of 1848. It is true that, long prior to that date, the government of the country had concerned itself from time to time, as occasions arose, with the more exceptional and formidable epidemic diseases, such as plague, cholera, and yellow fever; and that there were some rudiments of sanitary machinery, such as the now obsolete courts leet, and commissions of sewers, but with imperfect powers and confused and overlapping jurisdiction. Certain towns also had obtained private improvement Acts containing clauses of sanitary bearing, but, in the words of the Commissioners in 1845, "these local Acts appear to have been framed more with reference to the means of traffic in the streets and the general convenience of the inhabitants, than with any regard to their health." Shortly before 1848, a number of Acts had been passed containing model clauses on various subjects which might be adopted in any local Act. These were of advantage in facilitating the drafting of local Acts and in securing uniformity, but they have in some cases had the undesirable effect of stereotyping arrangements which, though considered suitable at that time, have been shown to be inadequate or objectionable by the light of advancing knowledge and subsequent experience. Thus the insufficiency of legal powers to compel water companies to supply wholesome water, is due to the circumstance that the Waterworks Clauses Act, 1847, by which the rights and duties of water companies in the provinces are regulated, was passed before it had been discovered that specific epidemic diseases could be propagated by the medium of polluted water.

The Public Health Act, 1848, may, however, be considered the first general Act relating to the public health; for though applying only where adopted locally or put in

force, it created a central health authority charged with the supervision of the health of the whole kingdom, and with power to act in any place where local circumstances called for action..

The way for this Act had been prepared in the previous decade: first by the fatal cholera epidemic of 1831-2, which had forcibly directed attention to questions of the public health, and secondly by two Acts passed in the fruitful legislative period which followed the passing of the Reform Bill of 1832, viz., The Poor Law Amendment Act of 1834, which improved and rendered more uniform the administration of the law relating to the relief of the poor, and created a central supervising board; and the Registration Act of 1837, which furnished an accurate statistical basis for enquiries respecting the number, distribution, and causes of deaths. A series of reports issued by the Poor Law Commissioners, largely the work of the late Sir E. Chadwick, had shown how great a proportion of the pauperism then existing was due to diseases and deaths from preventable causes, and had led to the appointment in 1843 of the Royal Commission on the Health of Towns, whose Report I have already quoted from. This report formed the basis of the Public Health Act of 1848.

The Commissioners advised "That new legislative measures, applicable to all towns and populous districts, are required for the introduction and maintenance, not only of an efficient and economical system of house-drainage and sewerage, paving and cleansing, in all towns and populous districts, but also for providing ample supplies of water for public and private purposes, and for the adoption of other means for promoting and securing the health and comfort of the inhabitants. That in all cases the local administrative body appointed for the purpose have the special charge and direction of all the works required for sanitary purposes, but that the Crown possess a general power of supervision. That upon representation being made by the municipal or other authority, or by a certain number of the inhabitants of any town or district, or part thereof, setting forth defects in the condition of such place as to drainage, sewerage, paving, cleansing, or other sanitary matters, the Crown direct a competent person to inspect and report upon the state of the defects; and, if satisfied of the necessity, have power to enforce upon the local administrative body the due execution of the law. That the local administrative authority have power to appoint, subject to the approval of the Crown, a medical

officer, properly qualified, to inspect and report periodically upon the sanitary condition of the town or district, to ascertain the true causes of disease and death, more especially of epidemics, increasing the rates of mortality, and the circumstances which originate and maintain such diseases, and injuriously affect the public health of such town or populous district."

The sanitary improvements upon the need for which most stress was laid by the Commissioners were sewerage and house-drainage. Other recommendations related to paving, scavenging, abatement of nuisances, regulation of offensive trades, sufficiency of water supply, width of streets, courts and alleys, cellar dwellings, privy accommodation, ventilation of public buildings, and regulation of common lodging-houses. The Commissioners did not see their way to make any recommendation with respect to open space at the back of houses, nor with respect to the ventilation of houses; and their only recommendation directly relating to infectious diseases was "that on complaint of the parish medical or other authorized officer, that any house or premises are in such a filthy and unwholesome state as to endanger the health of the public, and an infectious disorder exists therein, the local administrative body have power to require the landlord to cleanse it properly without delay; and, in case of his neglect or inability, to do so by its own officers, and recover the expense from the landlord."

Had the medical element been more strongly represented on the Commission—which contained only two medical members, viz., Sir Richard Owen, whose subsequent fame as a comparative anatomist may have thrown into the shade his earlier labours as a sanitarian, and Sir J. Ranald Martin — it is possible that more stress would have been laid on the need for directly opposing the spread of infectious diseases by measures of isolation and disinfection. But at that time medical knowledge concerning the propagation of fevers by specific contagia was less definite than it is now; and probably public opinion would not have sanctioned the expense of establishing isolation hospitals, or have approved any measures which savoured of interference with the liberty of the subject. It has doubtless been well for the country that the earlier efforts of sanitary reformers were directed towards promoting a condition of cleanliness unfavourable for the propagation of epidemic diseases, rather than towards combating these diseases by restrictive measures akin to

quarantine; but the course adopted has had the disadvantage that, when the question of hospital provision for infectious cases came to be taken up, it was done by the State for Poor Law purposes, and was thus started on a wrong track, as a measure for the relief of necessitous individuals rather than as one for the protection of the public health.

The Public Health Act, 1848, created a central board, the General Board of Health, with large powers, and authorised the formation in any place of a Local Board of Health, either upon the petition of not less than a tenth of the inhabitants or, where the average death-rate for a period of seven years exceeded 23 per 1,000, upon the initiative of the General Board of Health. It gave legal effect to the various recommendations of the Health of Towns Commission, and made the necessary financial and administrative provisions for carrying them out; and its clauses form in the main the basis of the Public Health Act, 1875, now in force. It applied, however, for the most part only in places where local boards of health had been formed, but one clause (§ 50) provided that in any parish or place containing less than 2,000 inhabitants the ratepayers in public meeting assembled might, by a majority of three-fifths, take steps for the cleansing, covering, and filling up of any pond, pool, open ditch, sewer, drain, or place containing, or used for the collection of, any drainage, filth, water, matter, or thing of an offensive nature or likely to be prejudicial to health; the making or improvement of a sewer, or the digging of a well, or providing a pump for the public use of the inhabitants. The work was to be done by the churchwardens, after approval at a second public meeting of ratepayers, at the cost of the poor-rate. The cumbrous procedure of this clause appears, however, to have been little resorted to in rural places.

The General Board of Health, as at first constituted, was a very able and vigorous body, but being too much in advance of public opinion, its constitution and powers were altered in 1854; and in 1858 it came to an end, its functions being divided between the Privy Council and the Home Office. This arrangement continued until 1871, when the Local Government Board was formed with the duty of supervising the administration both of the Poor Law and of the Sanitary Acts. The facilities for obtaining local self-government by the formation of local boards were extended by the Local Government Act of 1858,—too much so, indeed, for it became the fashion in some

parts of the country for mere villages or rural parishes to constitute themselves "urban districts" for the sake of escaping inclusion in a highway district, or of evading the carrying out of sanitary works. The last-named Act gave in urban districts a most important power, viz., that of making bye-laws with respect to the walls, foundations, etc., of new buildings for stability and the prevention of fires—subsequently extended to purposes of health—to the sufficiency of open space about buildings for the circulation of air, to the ventilation and drainage of buildings, and to water-closets, earth-closets, privies, ashpits, and cesspools in connection with buildings. Model bye-laws with respect to these matters were issued by the Home Office, and subsequently a more complete and explicit code has been framed by the Local Government Board. These bye-laws have been extensively adopted, and have probably done more than anything else to prevent the further creation in our towns of crowded slums and fever dens such as existed fifty years ago.

In rural districts, however, not only were the legal powers for the protection of the public health before 1872 far less ample than those of local boards, but such powers as existed were divided between two sets of authorities: viz., the "Nuisance Authority," under the Nuisance Removal Acts—that is to say, the Poor Law Guardians, and the "Sewer Authority," under the Sewage Utilization Acts —that is to say, the vestry in each parish; hence it is not surprising that but little was done before 1872 for the improvement of the sanitary condition of rural districts. The nuisances for the abatement of which powers were given by the Nuisance Removal Act, 1855, appear to comprise only things which stink; but the definition was subsequently enlarged so as to include overcrowding, ill-ventilated workplaces, and the emission of black smoke from chimneys other than those of dwelling-houses.

Other Acts passed during the period relate to the provision of burial grounds, and of public baths and washhouses, and the regulation of common lodging-houses.

Hitherto, the only Acts for the direct combating of infectious disease by such measures as disinfection and the provision of hospital accommodation and medical attendance were the Disease Prevention Acts, very inappropriately so named, since they were only to be put in force by the central authority when some part of the country was already affected by a formidable epidemic, and in practice were put in force only in the case of an exotic disease such

as cholera. The Sanitary Act of 1866 made an important extension of the scope of the law in this direction. It empowered the provision of hospitals or temporary places for the reception of the sick, and the removal thereto under order of a Justice of any person suffering from any dangerous infectious disorder who was without proper lodging or accommodation, or was in a room occupied by more than one family, or on board any ship or vessel. It also empowered the provision of an ambulance for the removal of the sick, and of a place for the disinfection of woollen articles, clothing, or bedding which had become infected, and the compulsory cleansing and disinfection of infected houses, and articles in them likely to retain infection. It forbad the exposure of an infected person in any public place, or the use by him of any public conveyance without notice to the driver; also the giving, lending, selling, or exposing of infected articles, and the letting of infected houses or rooms.

These powers, however, being conferred some upon "nuisance authorities" and others upon "sewer authorities," were little, if at all, used in rural districts where the "nuisance authority" and the "sewer authority" were different bodies of men.

In 1869 a Royal Commission was appointed to inquire into and report upon the operation of the sanitary laws in England and Wales (except London) so far as they apply to sewerage, drainage, water supply, removal of refuse, control of buildings, prevention of overcrowding, and other means of promoting public health, and for preventing the introduction and spreading of contagious and infectious diseases, and epidemics affecting the health of man; also into the constitution of the central and local authorities charged with the administration of these laws, and the formation of proper areas, and into that part of the registration system which relates to certificates of causes of death, and to suggest improvements in all or any of these matters.

The outcome of the report of this Commission was the passing of the Public Health Act, 1872, which marks a second important epoch in the public health history of this country. Hitherto, the progress of sanitary improvement had been fitful and partial, and had been confined to the towns, the rural districts being little if at all affected by it; but from this date there has been, speaking generally, a steady advance all along the line, though of course all places have not participated in this advance to an equal extent.

The Act of 1872 created two classes of inland sanitary authorities, viz., urban and rural, the Urban Sanitary Authorities being the Town Councils of boroughs, the Improvement Commissioners in districts under local Acts, and the Local Boards in other towns, the Rural Sanitary Authorities, being the Boards of Guardians in whom were vested the powers both of "nuisance authorities" and "sewer authorities" under the earlier Acts. The sanitary powers of the "parish," more often used to obstruct than to promote sanitary improvement, thus ceased, except where delegated by the Rural Sanitary Authority to parochial committees; but certain limited sanitary functions have recently been assigned to parish councils under the Local Government Act of 1894. There were also formed Port Sanitary Authorities, consisting of representatives chosen by the riparian authorities.

Each of these several authorities was now obliged to appoint one or more medical officers of health; whereas previously only urban authorities had the power to make such an appointment, and it was optional upon them whether they did so.

The codification of sanitary law in the Public Health Act, 1875, greatly aided sanitary administration, and further powers with respect to the notification and prevention of infectious diseases, the provision of isolation hospitals, and other sanitary purposes, have been conferred by more recent Acts; while the urban and rural sanitary authorities, under the name of District Councils, have been placed on a more popular elective basis.

The creation, in 1888, of County Councils as a supervising body in each county has tended to stir up the more sluggish local authorities to a better performance of their duties, more especially in counties where the County Council has exercised its power of appointing a county medical officer.

In London, a system of local authorities, in the form of elective vestries, was created by the Metropolis Management Act of 1855; and for purposes affecting the Metropolis as a whole, the same Act also formed the Metropolitan Board of Works, a body to whom London owes its main drainage system and the embankment of the Thames. This Board was superseded in 1888 by the London County Council, and London sanitary legislation, which had lagged behind that in force in the provinces, was extended and brought up to date by the Public Health (London) Act, 1891.

The provision of isolation hospitals in the Metropolis

still rests however with a Poor Law body; although since 1889 the hospitals of the Asylums Board have been available for persons other than paupers, and their use no longer involves the disqualifications attaching to the receipt of Poor relief.

One other important means for the prevention of disease is also still in the hands of the Poor Law authorities, both in London and the provinces: I allude to vaccination. The earliest step taken by the State for the promotion of vaccination was the foundation, early in the century, of an establishment for the gratuitous distribution of vaccine lymph. In 1843, Boards of Guardians were authorised to contract with medical practitioners for the gratuitous performance of vaccination. In 1853, on the representations of this Society, an Act was passed rendering vaccination obligatory, but no machinery was appointed for enforcing the duty. This omission was supplied by the Vaccination Act of 1869, and the Act of 1871 rendered compulsory the appointment of an officer charged with the duty of enforcing the Vaccination Acts. Of late years, however, owing to an organised opposition to vaccination, its enforcement by the Guardians has ceased in many parts of the country, and the number of vaccinations has greatly fallen off; of the children whose births were registered in 1894, the last year for which figures are available, the proportion not finally accounted for as regards vaccination was 19.5 per cent., viz., 20.6 per cent. in London, and 19 per cent. in the provinces. This is the highest proportion in any year since 1872, the figures in previous years having been as low as 5.7 per cent. in London in 1881, and 3.8 per cent. in the provinces in 1875. In the recent Session of Parliament an Act has been passed relieving the "conscientious objector" to vaccination, and superseding the system of arm-to-arm vaccination, which has hitherto been recognised, by one of domiciliary vaccination with glycerinated calf-lymph. How far this change will conciliate the opponents of vaccination, or secure a larger percentage of vaccinated children, it is as yet too soon to judge.

We may now glance briefly at the chief sanitary improvements which have been effected as the outcome of the legislation of which I have given a summary.

At present all the larger towns of the country, and most of the smaller ones, are more or less completely sewered; the chief exceptions being some Kentish towns, in which

cesspools leaking into the chalk afford an only too easy method of getting rid of sewage; and some towns in the coal-mining districts, where difficulty arises through the subsidence of the ground which takes place after coal-getting. The disposal of sewage is a problem still not solved; subsidence, filtration and irrigation, chemical, electrical, and biological methods have all been tried, and not a year passes without some new method being brought forward; but the hopes formerly entertained of making a profit out of it have been doomed to disappointment, and all that is now aimed at is to render it innocuous at a moderate cost. Some steps have been taken to purify our rivers, but much more remains to be done in this direction. In proportion, however, as rivers become disused as sources of domestic water supply, their pollution, however objectionable on æsthetic grounds, loses a good deal of its importance from a health point of view. Even if all sewage could be excluded from our rivers they would still, in all but the uppermost portions of their course, be too much exposed to pollution of other kinds, such as manufacturing refuse, muddy water off roads, surface washings off manured fields, decaying vegetable matter, droppings of cattle, and sheep washing, to be a proper source of drinking water, at any rate without thorough filtration.

The construction of house drains (though of course scamped work is still only too frequently met with) has been greatly improved, especially since the introduction of the water test as a means of ascertaining their imperviousness. Their ventilation, efficient trapping, and severance from direct communication with the interior of houses, are important features of modern drains.

A noteworthy result of efficient drainage, coupled with the enforcement of proper building regulations, is that well-administered towns become healthier as they increase in size, since the central and closely-built parts are converted into business quarters, and the population is transferred to new suburbs, well laid out and built on virgin soil, which, where sewerage has been constructed when building first commenced, has never been impregnated with the soakage from privies and cesspools.

The earlier sanitary reformers, impressed by the evils accruing from the accumulation of human excrement in privies and cesspools in the immediate neighbourhood of dwellings, strongly urged its removal through the sewers by means of a water-carriage system. At that time, however, not only was the construction of sewers and drains

imperfect, but the proper details of the closet arrangements had not been worked out, nor were people of the working-class educated to the use of water-closets. In consequence, evils such as blocked drains, filthy pans, and escape of foul air into houses, were of frequent occurrence, and a further danger was the liability of foul matter being sucked up into the water pipes through their direct connection with the closet pans. Hence, some thirty years ago, there was a reaction in favour of the so-called "dry" or "conservancy" methods, by which excrement was stored up for more or less prolonged periods in the neighbourhood of houses. At the present time, however, these conservancy methods are being rapidly superseded in our towns by the water-carriage system. In the towns of the South of England the latter method is now nearly universal, and the Midland and Northern towns are getting dissatisfied with their midden privies and pail closets, and are coming to favour the more cleanly method on economical as well as on sanitary grounds. It is found that the collection and carriage of night soil does not pay, as in the present state of agriculture there is little or no demand for it; and it has to be got rid of by burning in destructors, by barging it out to sea, or by tipping it into quarries and waste places. As an abstract principle we may regret that so much potentially valuable fertilising material should be wasted: and should the failure of our wheat supply for want of fixed nitrogen, which Prof. Crookes foresees, ever become imminent, the excremental refuse of our large towns may acquire a market value sufficient to pay for its removal in the dry way with the frequency demanded in the interests of health; but under present circumstances its economic value certainly does not outweigh the sanitary disadvantages attaching to its storage and collection by any methods hitherto found practicable in such districts.

As regards water supply there is not one of our large towns, and there are few of the smaller ones, which have not a public water service, and shallow wells in towns have fallen into more or less complete disuse as a source of drinking water, especially where the public supply belongs to the local authority. Much pains and expense have been incurred to secure water from pure sources, as deep wells and upland streams and gathering grounds, in place of supplies taken from polluted rivers; some towns, as Liverpool, Manchester, and Birmingham, having brought water at vast cost from distant mountain regions. Where, as in the

case of London, the supply continues to be taken from rivers, the need for efficient filtration is recognised.

As regards means of combating infectious diseases, the notification of such diseases is now compulsory in districts having an aggregate population, according to the 1891 census of nearly twenty-eight out of the twenty-nine millions in England and Wales.

The provision of hospitals for the isolation of infectious diseases has made great progress in recent years. Besides London, the needs of which in this respect are supplied by the Metropolitan Asylums Board, I find from a Parliamentary Return, published in 1895, that out of 1,653 sanitary authorities, urban, rural, and port, 631 had up to the end of 1892 made, alone or jointly with other authorities, some sort of provision for the isolation of infectious cases. Since that date the establishment of isolation hospitals has been much extended, 216 loans for hospital purposes—not all, however, relating to different hospitals—having been sanctioned by the Local Government Board in the five years 1893-7; while many other authorities have provided themselves with accommodation without the aid of loans. The use of hospitals has also become much more general, as the prejudice with which they were at first popularly regarded has disappeared with experience of their benefits, and as their use has been made free without charge; so that in some districts a very large percentage of the cases of diseases, such as scarlet fever, are now isolated in hospital. The use of steam for disinfecting articles of clothing and bedding has also extended. Time would fail me to allude to the many other directions in which a care for the public health is manifested, either by the provision of appliances for promoting and safeguarding it, or by the supervision and regulation of places and processes which may involve danger to it.

In rural districts, especially in the unprogressive parts of the country, less has been done in the way of sanitary improvement than in the towns: partly because in such districts public opinion is less advanced; partly because in a more sparse population collective systems of drainage and water supply are less needed than in a closely aggregated one; and partly also on account of the financial difficulties arising from agricultural depression, and from the want of power in the present state of the law to apportion the cost of sanitary improvements upon the part of a parish benefited by them. Still, in most rural districts more or less supervision is exercised over matters affecting

the public health, and detailed improvements are made as required. Urban powers, too, have of late years been more frequently granted and exercised in the more populous rural districts.

Turning now to consider the results which have followed the changes of which I have spoken, we find in the first place that, notwithstanding the adverse effect of the increasing aggregation of population in towns, there has been during the past fifty years a great diminution in the death-rate from all causes.

The average annual death-rate per 1,000 persons living, which was 23.1 in the five years 1847-51, was in the period 1892-6 only 18.1 : a saving of life of five persons annually in every thousand, or 145,000 in the population of England and Wales as enumerated at the census of 1891.

The diminution of the death-rate has been greater in the towns than in the country districts, it having fallen in the former from 24.7 in the ten years 1851-60 to 20.3, in the ten years 1881-90, and 18.8 in the five years 1892-96; while the rural death-rate fell from 19.9 in the ten years 1851-60 to 17.3 in the ten years 1881-90, and 16.7 in the five years 1892-96. The average death-rate in the thirty-three great towns of England and Wales in the five years 1892-96 (20.0) is almost as low as that (19.9) in the country districts in 1851-60.

That this reduction in the death-rate is due in the main to sanitary improvement rather than to improved social conditions appears probable when we follow its course in five-yearly periods. In the period 1847-51, a period marked by epidemics of cholera and influenza, as well as of fever (largely typhus), the death-rate was 23.1. In the next five years, corresponding to the brief but vigorous reign of the first General Board of Health, it fell to 22.4, but notwithstanding the general prosperity of the country it remained almost unaltered at this point for twenty years. In the five years, however, which followed the passing of the Public Health Act, 1872, it fell from 22.3 in the preceding period to 21.6, and there has been a steady decline in each successive quinquennium since then to 18.1 in the five years ending 1896 (see Chart I). This conclusion as to the reason for the decline in the death-rate is strengthened when we examine the tables of the Registrar-General with a view to ascertain from what causes the deaths have diminished. It will be found that this has been largely in the diseases of the " zymotic " class, and in this class, among

those diseases which are most amenable to preventive measures in so far that their natural history is best known, and that they are popularly esteemed of sufficient gravity to warrant the expenditure of money and the taking of pains for their prevention. Thus the death-rate per million inhabitants from small-pox fell from 312 in the period 1847-52 to 12 in 1887-91. The death-rate from continued fevers fell from 1,184 in 1847-51 to 184 in 1892-96; and that from "cholera" from 660 in 1847-51 to 23 in 1892-96, while that from scarlet fever declined from 1,001 in 1862-66 to 118 in 1892-96. The mortality from continued fevers, like the general death-rate, fell between the quinquennium 1847-51 and 1852-56, but then remained at nearly the same height until 1872, since when it has rapidly declined (see Charts II, IV and V).

The lessened mortality of recent years has not, however, been shared equally by persons of all ages; the gain has been in the earlier periods of life, and a smaller proportion of persons live to extreme old age at the present time than did half a century ago. More persons reach adult age now than formerly; but of those who reach middle age the expectation of life is, *cæteris paribus*, somewhat less than it was fifty years ago. In a comparison of several successive life tables, given in the Supplement to the 55th Annual Report of the Registrar-General, it is shown that in England and Wales the mean expectation of life at birth for males was, in the period 1838-54, 39.91 years; in 1871-80, 41.35 years; and in 1881-90, 43·66 years. In certain selected healthy districts it was in the period 1849-53, 48.56; and in 1881-90, 51.48 years. Females live longer; their mean life-time in England and Wales was in 1838-54, 41.85 years; in 1871-80, 44.62 years; and in 1881-90, 47.18 years. In the standard healthy districts it was in 1849-53, 49.45 years, and in 1881-90, 54.04 years. Thus, in each case there was in 1881-90 an increased expectation of life at birth ranging between 3 and 6 years. But at ages from 27 years onwards in the case of males, and from 45 years onwards in the case of females, the mean expectation of life was less in 1881-90 than in 1838-54. Even in the standard healthy districts there is a similar diminished expectation of life in 1881-90 as compared with 1849-54, though the diminution begins to occur later than in the kingdom generally, viz., at 32 years in the case of males, and at 48 in the case of females. We may infer that while the sanitary improvements of the past half century have diminished the fatality of the infective diseases of earlier life, they have done

little to postpone the degenerative changes of later life; and that though the present conditions of life are favourable to the survival of the young, the increased wear and tear of our strenuous modern life, especially in towns, bring about an earlier final breakdown of the vital machinery. Moreover, it is probable that a number of weakly lives, which under the less favourable conditions of fifty years ago would have been extinguished in childhood, are now enabled to survive into adult life, though they have not sufficient vitality to reach old age.

Looking at the Registrar-General's table, which shows the average annual death-rates from various causes in successive groups of years, we find that while the deaths from old age have diminished from 1,352 per million living in 1861-65 to 929 per million in 1891-95, on the other hand, during the same period those from cancer have increased from 368 to 712 per million; those from diabetes from 29 to 69 per million; those from diseases of the circulatory system from 996 to 1,677 per million; those from diseases of the urinary system from 246 to 453 per million; and, what is probably of a significance greater than the actual figures, those from intemperance have increased from 41 to 68 per million.

The infant mortality shows, unfortunately, but little diminution during the fifty years under review. The deaths of infants under one year old out of 1,000 born, were 156 in the five years 1847-51, and remained at about the same figure till 1876. In the period 1877-81 the infant mortality fell to 141 per 1,000 born, but has since again risen to 151 in the years 1892-96. Among the principal causes of death in infants under one year are measles and whooping-cough; diarrhœa and enteritis; tabes mesenterica, and other tubercular affections; premature birth, convulsions and dentition; bronchitis and pneumonia; debility and atrophy. From some of these causes, as premature birth, the mortality is increasing; from others, as convulsions and dentition, the apparent mortality is decreasing, perhaps mainly as the result of more careful and definite certification.

Changes of practice and fashion iu the certification and classification of diseases have indeed an influence not to be ignored in the apparent increase or diminution of the mortality from particular diseases. In recent years, with the advance of medical knowledge there has been increased accuracy and definiteness in certifying the cause of death; and more especially since the Registrar-General has adopted

the practice of asking by letter for supplementary information concerning deaths from vaguely certified causes. Hence there has been a great diminution in the number of deaths certified from ill-defined causes, such as dropsy, tumour, abscess and mortification, and from causes such as convulsions, of which the certification is based on a prominent symptom rather than on the essential nature of the disease. This tendency will have to be borne in mind in considering the mortality from certain epidemic diseases, as diphtheria, diarrhœa, and fever. Of the epidemic diseases with which we as a Society are especially concerned, we find that during the fifty years under review the mortality from continued fever and cholera, disease against which sanitary efforts from the first have been specially directed, has steadily declined throughout the period. In the case of other diseases, as scarlet fever and diarrhœa, the mortality rose to a maximum about the middle of the period, and has since declined to below what it was at the commencement. Whooping-cough and measles show little or no reduction; while the mortality from diphtheria and influenza, after having declined, has increased in recent years. Phthisis and other tubercular diseases have declined.

We may consider in a little more detail these several diseases as factors in the mortality of the past fifty years.

Cholera was epidemic in the years 1847 and 1848, and again in 1853-54. It is an oft-told tale how, in South London, it fell with different incidence upon the houses supplied by the two water companies whose mains ran side by side, and how the customers of one company who suffered the more in the earlier epidemic, suffered the less in the second epidemic, the company having in the meantime removed its intake higher up the river and improved its filtration. In 1866 epidemic cholera again occurred, and again it was found in East London to be associated with the distribution of water polluted by specifically infected sewage. Since 1866 Asiatic cholera has never become epidemic in England; for though during recent decades it has been epidemic in neighbouring European countries, and cases have been imported from time to time, yet either they have been arrested by the system of port sanitary defence organised in recent years, or if they have passed it, the seed has not fallen in a congenial soil, and the disease has died out without propagating itself. The only exception was in 1893, in which year epidemic cholera gained some footing in Grimsby—a town in which enteric fever

and diarrhœa are endemically prevalent—and from thence a certain number of cases of cholera were disseminated over neighbouring counties, largely through the agency (as there is reason to believe) of shell-fish taken from the sewage-polluted tidal waters. Apart from epidemic years, a certain number of deaths are returned from "cholera" year by year, their number varying with that of the deaths from "diarrhœa;" and they represent a severe form of that disease, rather than true cholera (see Chart II).

From "Fever" the death-rate has very markedly declined between 1847 and the present time, and since 1866 with a continuous steady fall. In 1847 the distinction between typhus and typhoid fevers had already been pointed out by Dr. A. P. Stewart, and it was confirmed by Sir W. Jenner in 1849. It was not, however, made in the Registrar-General's classification until 1869; and even after that date the different kinds of fever were but imperfectly distinguished by the older generation of medical men. Hence, among the deaths ascribed to typhus and to simple and ill-defined fevers in the earlier years after 1869, there were probably many which should have been ascribed to enteric fever; and the more rapid diminution in the number of deaths from the first-mentioned forms of fever than from enteric fever, is partly due to the transference of cases from the two former categories to the latter as medical diagnosis improved (see Chart III).

Typhus is known to have been very prevalent in England in 1847 and 1848, the years immediately following the failure of the potato crop in 1846, which drove a large number of destitute Irish to this country. With the regulation of common lodging-houses, the closing of cellar dwellings, and the improvement of the dwellings and of the general condition of the working classes, it has steadily declined, until it is now so rare a disease that comparatively few medical men have ever seen it. It is still met with among the colonies of squalid Irish congregated in our large towns, especially in Lancashire, but when it breaks out elsewhere its spread is generally due to the failure to recognise at the outset a disease so unfamiliar.

Enteric fever, on the other hand—though its mortality has also declined, and in 1892-96 was less than half wha it was in 1869-71—is much more frequent and generally distributed than typhus, and is now not much more fatal in the large towns than in the rest of England and Wales. Generally speaking, it is more prevalent in the north than in the south of England, the counties in which it has occa-

sioned the highest mortality during the past ten years being Durham, Yorkshire, Lancashire, Nottinghamshire, South Wales, Cheshire, Northumberland, and Staffordshire. These counties contain, besides large towns, many populous mining and manufacturing places of recent growth, in which drainage, water supply, and sanitary administration generally are as yet very imperfect, and in which the midden privy still prevails in all its primitive nastiness.

It is somewhat difficult to appraise the respective effects of the several measures by which the prevalence of enteric fever in our towns has been lessened; but the most potent, probably, is the introduction of a supply of pure water to replace a contaminated supply from wells and other local sources. Many instances could be quoted in which towns in which enteric fever was endemic have become practically free from it on the introduction of a supply of water from an extra-mural source. On the other hand, a public water service, should it become specifically contaminated, gives opportunity for an epidemic of enteric fever, on a scale of magnitude and with a rapidity of development far exceeding what was possible when the water supply was obtained from a number of different sources.

The following are some instances in recent years of typhoid epidemics of water origin in towns ordinarily comparatively free from that disease.

Town.	Population, estimated.	Duration of Date of Epidemic.	Cases.	Deaths.	Rates per 1,000 Inhabitants.	
					Cases.	Deaths.
Bangor ...	15500	1882. 3½ months, May-Sept.	548	42	35.3	2.7
Beverley ...	11425	1884. 2½ months, June-Aug.	231	12	20.0	1.2
Kidderminster	25500	1884. 3½ months, Aug.-Nov.	1200	89	47.0	3.5
Worthing ...	19290	1893. 7 months, May-Nov.	1411	186	73.0	9.6
Newport, I. of Wight	13533	1894. 3½ months, Aug.-Dec.	516	57	38.1	3.77
Maidstone ...	33830	1897. 2 months, Sept.-Oct.	1847	132	54.6	3.9

Next in importance to a pure water supply in the reduction of enteric fever prevalent in our towns is the prompt conveyance of excretal matter out of them, instead of storing it up in their midst. Broadly speaking, it may be stated that water-closet towns have a lower typhoid mortality than privy-midden towns. According to figures collected by Dr. Scurfield, Medical Officer of Health for Sunderland, in the six years 1890-95, the death-rates per 1,000 inhabitants from "fever", in thirty-one privy-midden towns, averaged 0.26, while in twenty-five water-closet towns they averaged 0.15. The corresponding rates for diarrhœa were 1.08 and 0.52 respectively in the two classes of towns. It has also been found in many instances that in the same town the incidence of enteric fever is greater on houses with privies than on houses with water-closets.

Further light on the cause of the endemic prevalence of enteric fever in certain towns has been thrown by recent researches, which show that the Bacillus typhosus is capable of long retaining its vitality in porous earth containing an excess of organic matter. This may help to explain how it is that in towns of which the site and subsoil have been allowed to become sewage-sodden, the carrying out of sanitary works does not always at once reduce the prevalence of enteric fever. In the same way the drainage of the fens did not at once stamp out ague, but the disease gradually declined, until it is now almost extinct.

Diarrhœa has in its epidemiological aspects several points of similarity to enteric fever. Both diseases attain their greatest prevalence in the late summer and autumn, though earlier in the case of diarrhœa than in that of enteric fever, and the years which are marked by an excessive mortality from the one disease are usually marked by an excessive mortality from the other also; as was the case in 1893 and 1895, and probably also in 1849, 1852, 1854, 1857, and 1865, in which years the mortality both from diarrhœa and "fever" was excessive, though at that time the several forms of fever were not separately classified. In some towns also, as at Nottingham and Grimsby, Preston and Sunderland, both diseases prevail endemically, though in others no such concurrence is observed. Again, epidemics of enteric fever have not unfrequently been preceded by an unusual prevalence of diarrhœa; this was the case with those at Kidderminster and Maidstone, mentioned in the table previously given. But while the mortality from enteric fever has greatly and almost con-

tinuously declined during the last fifty years, that from diarrhœa fluctuates greatly from year to year; and though it has, on the whole, declined since the period 1867-71, the diminution has been much less in proportion than that of enteric fever (see Chart II). Indeed, it may be doubted whether the apparent decrease in recent quinquennia is not mainly due to the transference of deaths from the heading of "Diarrhœa" to that of "Enteritis." The deaths under the latter heading have greatly increased in recent years; having been at the rate per million inhabitants of 108 in 1877-81, 118 in 1882-6, 156 in 1887-91, and 270 in 1892-6. There is an objection on the part of some medical men to the use of the word "diarrhœa," partly (especially in health resorts) on the ground that the deaths from diarrhœa are reckoned in the "Zymotic mortality," to the discredit of the district; partly on the ground that diarrhœa is a symptom of many diseases, rather than a disease in itself. There can, however, be no doubt that the summer diarrhœa which causes such a heavy mortality among infants, especially in certain towns, is a definite disease owning a specific cause; and unless a better term can be found, and its universal acceptance secured, a change from the established nomenclature is to be deprecated.

The mortality from diarrhœa takes place mostly in the third quarter of the year, and varies very closely with the mean temperature of that quarter, as is shown by the following Table:—

Period.	1852-56.	1857-61.	1862-66.	1867-71.	1872-76.	1877-81.	1882-86.	1887-91.	1892-96.
Average diarrhœa mortality per million ..	944	905	844	1111	985	761	724	581	646
Mean temperature of 3rd quarter. Departure from average of 48 years ..	-0.1°	+0.5°	-0.5°	+1.2°	+0.7°	-0.4°	-0.1°	-1.2°	+0.3°
Years above average ..	2	4	1	4	5	2	2	1	3
Years below average ..	3	1	4	1	..		3	4	2

The physical conditions conducing to the occurrence of epidemic diarrhœa, so far as known, are a porous soil impregnated with organic matter and with a water level not far below the surface, and a high ground temperature, viz., of 56° F. and upwards at a depth of 4 ft. The improper artificial feeding of infants also greatly increases their liability to the disease: it is comparatively rare for

an infant wholly fed at the breast to die of diarrhœa. Assuming the poison to be generated in the soil, the mode in which it gains access to the human body, whether by ground air, by dust, or by tainting the food, remains to be discovered. The disease is pre-eminently one of towns, and the comparatively small effect which sanitary improvements have had in reducing its mortality is, perhaps, attributable to the increasing preponderance of the urban over the rural population, and to the increased use of artificial foods of various kinds in place of breast milk in the feeding of infants.

Diphtheria first appears under a separate heading in the Registrar-General's Reports in the year 1855, though the disease had previously been known under other names, and had been prevalent in different parts of England and Wales in the early fifties. It was epidemic in 1858 and 1859, and caused a mortality in those years respectively of 339 and 517 per million: figures higher than have been since reached. After 1859, the diphtheria death-rate steadily declined to 121 per million in the five years 1867-71, and remained at that level until 1881. It then rose again to 318 in the year 1893, and has remained not far below that point in subsequent years, the mean rate during the five years 1892-6 having been 277 per million (see Chart IV). The distribution of diphtheria, as between town and country, has also varied: during the past fifteen years, from being a disease especially of the country districts it has become a disease of the towns, and more especially of London, as shown by the following Table:—

Diphtheria Death-rates per 1,000 *Inhabitants.*

Period.	England and Wales.	Great Towns.	Rest of Kingdom.	London.	Other Great Towns.
1882-86	.16	.16	.16	.23	.11
1887-91	.17	.22	.15	.32	.14
1892-96	.28	.36	.23	.60	.20

It is, as a rule, more prevalent in the south of England than in the north; and it often happens that healthy districts suffer severely from diphtheria, while unhealthy towns having a high fever, diarrhœa, and general death-rate, and a large infant mortality, may nevertheless escape diphtheria lightly. A town heretofore comparatively free

from diphtheria may develop a sustained prevalence of that disease, which may continue for several years, and then subside to its former low level.

The cause of the increased mortality from diphtheria in recent years is very obscure. There has, indeed, probably been to some extent a transference of deaths from the headings of croup and quinsy and sore throat to that of diphtheria; but the falling-off in the deaths certified from the former diseases is insufficient to account for the increase in those from the latter disease. There can be no doubt that diphtheria very frequently spreads through the agency of schools; but against the view that the compulsory school attendance established under the Elementary Education Act, 1870, is the main cause of the recent recrudescence of diphtheria, is to be set the circumstance that the increase of diphtheria only commenced about 1882, or some twelve years after education became compulsory.

We may hope that in the future bacteriology will help to solve the puzzling problems connected with this disease. Already it has thrown light on its observed behaviour, by showing that the diphtheria bacillus may be present in the fauces of a patient after apparently complete recovery, in those of a person with a sore throat not presenting the clinical features of diphtheria, and even in those of a person attending on diphtheria cases, but whose throat may appear perfectly healthy.

Scarlet fever in its clinical features is so nearly allied to diphtheria that the two diseases were grouped together by the Registrar-General until 1855, and they have sometimes even appeared to be interchangeable—at any rate, diphtheria is not unfrequently engrafted on scarlet fever—yet in their local distribution, and in the history of their prevalence since 1855, the two diseases are in strong contrast: for while diphtheria is most fatal in London and in the agricultural counties of the south and east of England, scarlet fever causes the highest mortality in the north and north-west. and especially in the densely-peopled manufacturing and mining districts of South Wales, Lancashire, the West Riding, and Durham. The causes of this difference of distribution cannot be assigned; but I may mention two conditions which I have often observed in districts which have have suffered severely from scarlet fever, and which are more general in the manufacturing and mining than in the agricultural districts, viz., (1) that the houses have behind them large common yards or back streets, which afford a meeting-place for the children of different households; and

(2) a foul condition of the ground surface about houses, from lodgment of slop-water and littering about of refuse, coupled with defective paving.

The most remarkable feature in the history of scarlet fever during the last half century has been the great diminution of its fatality in the later years of the period. In successive periods of five years, from 1847-51 to 1867-71, scarlet fever exhibited an increase of fatality (especially considering that in the earlier periods the figures for scarlet fever had included also the deaths from diphtheria), attaining its maximum in the ten years 1862-71, the highest mortality being in the years 1863, 1864, and 1870. But since then it has steadily declined, so that the average annual death-rate living, which was 1,014 per million persons in the period 1867-71, was only 188 per million in 1892-96. Moreover, while in the earlier part of the half century formidable epidemics of scarlet fever affecting the whole kingdom recurred at intervals of four, five, or six years, the later years have shown no such general epidemics: local epidemics there are still, but in any given year they affect only particular localities and not the country generally; so that the curve of scarlatinal mortality for the whole country presents a nearly uniform and steady decline, in place of the violent fluctuations which affected it during the earlier periods (see Chart IV).

The diminished mortality from scarlet fever is to be attributed to the diminished virulence rather than to the diminished prevalence of the disease. It is a matter of common observation among medical men, whose memory carries them back so far, that scarlet fever is now a comparatively mild affair, very different from the formidable disease which it was twenty-five or thirty years ago. Among cases admitted into the hospitals of the Metropolitan Asylums Board, the proportion of deaths in the five years 1872-6, was 12.5 per cent.; in 1877-81, 12.7 per cent.; in 1882-86, 10.7 per cent.; in 1887-91, 8.3 per cent.; in 1892-96, 5.6 per cent.; and in 1897, 4.07 per cent. Among cases of scarlet fever notified in London the mortality fell from 5.6 per cent. in 1890 to 3.4 per cent. in 1897; being 4.8 per cent. in the four years 1890-93, and 4.0 per cent. in the four years 1894-97. While the notifications in London fell only from 5.3 per 1,000 living in 1890-93, to 5.0 per 1,000 in 1894-97, the deaths from scarlet fever fell in greater proportion, viz., from .25 to .18. How far the milder type which scarlet fever has assumed in recent years may be due to improved sanitary conditions it is

difficult to say; but it may be noted that in recent years the disease has in some instances exhibited something of its old deadliness, when it has become epidemic in districts in which filth conditions were allowed to prevail: thus, in a Nottinghamshire colliery town, in 1893, scarlet fever caused a death-rate of 5.3 per 1,000 inhabitants; and in an epidemic in a Lincolnshire village in 1894, 10 per cent. of the notified cases were fatal.

It is doubtless owing to the recollection of its former severity that the public have a wholesome dread of scarlet fever—greater, indeed, than its present mildness might otherwise inspire—and are therefore willing to take some trouble and incur some expense in adopting measures of isolation and disinfection for the purpose of preventing its spread. It is notifiable, and it forms the great bulk of the cases treated in isolation hospitals. In London, in 1897, 67 per cent. of the notified cases of scarlet fever were isolated in the Asylums Board's hospitals, and in some towns the proportion has exceeded 90 per cent. The greatest number of deaths from scarlet fever is in the third year of life, whereas the greatest number of deaths from whooping-cough takes place in the first year, and from measles in the second year of life. But the later maximum, in the case of scarlet fever, is due to the greater liability to contract the disease in years after the first; of those who do contract it, the mortality is greatest in the first year of life, and rapidly diminishes with each succeeding year. Thus, among cases admitted into the Metropolitan Asylums Board's Hospitals in the six years 1892-97, the mortality per cent. was:—

Under 1 Yr.	1 to 2.	2 to 3.	3 to 4.	4 to 5.	5 to 10.	10 to 15.
25.8	20.4	15.2	11.3	7.4	3.0	1.2

After 15 it again rose slightly.

A postponement of the average age at which children contracted scarlet fever would therefore result in a marked reduction of the mortality therefrom, even if the prevalence of the disease were in no degree diminished, and if as large a proportion of persons eventually contracted it. In one respect scarlet fever lends itself to preventive measures, in that it usually commences suddenly with well-marked symptoms; though, on the other hand, infection is long retained, and it happens only too often that a convalescent infects others after his apparent recovery.

Measles and whooping-cough have unfortunately undergone no such diminution as scarlet fever, and at the present

time each of them causes a greater mortality than any other disease of the "zymotic" class except diarrhœa. The death-rate from measles is as high now as fifty years ago; and though that from whooping-cough has undergone a slight diminution in the past twenty years, this may be partly explained by the diminution of the birth-rate: whooping-cough being most fatal to infants in the first year of life, of whom there would be fewer with a reduced birth-rate. If we examine the statistics of a single populous city, such as London or Manchester, we find that measles is epidemic about every other year; but as the epidemics in different parts of the country are not synchronous, the curve for the whole kingdom is much more uniform: there are, however, waves of increased mortality at intervals averaging about four years, and waves of longer period, rising and falling in about twenty years (see Chart IV). The undiminished mortality from these diseases may be explained by the increased aggregation of the population in towns, which has counterbalanced the influence of improved sanitary conditions; both of these diseases being most fatal in the large urban districts. Hitherto, but little success has attended such efforts as have been made by local authorities to attack them directly: partly because of the apathy with which these diseases are popularly regarded, partly on account of their both being infectious at an early stage before the distinctive symptoms, viz., the rash and the characteristic cough, appear. Compulsory notification has been tried, but has been given up in many places where it had been adopted. Hospital isolation has been found impracticable, owing to the large number of cases for which accommodation would have to be provided, and to the tender age of many of the patients. We must hope that some day the popular conscience will be roused to consider it a duty at least to avoid exposing children to the infection of these diseases, and to take proper care of them if they should be attacked. Even more than in the case of scarlet fever, every year that a child passes without contracting measles or whooping-cough diminishes the likelihood of its dying of them; and the low mortality from these diseases among classes of society in which the patients receive proper attention, shows that the complications to which a fatal issue is generally due are largely preventable.

Small-pox, unlike the last-named diseases, is one which inspires popular dread, and consequently much attention has been devoted to its prevention, and when it occurs vigorous means are taken to stamp it out. As a

result, the mortality from this disease, in England and Wales, has greatly declined during the past fifty years, viz., from 312 per million in 1847-51, to 12 per million in 1887-91, since when it has slightly risen; but the regular decline of the disease was greatly interrupted by the formidable and world-wide epidemic of 1870-72 (see Chart V). The means which have been credited with bringing about this reduction are: 1st, the general improvement of sanitary conditions; 2nd, vaccination; and 3rd, isolation and disinfection. Without entering into the controversy which has raged around these topics, I may point out that there are statistical grounds for thinking that each of these factors has had its influence. In view of the great reduction in the prevalence of typhus, a disease spread much in the same way as small-pox, we can hardly believe that the improvement of the housing of the poor, the regulation of common lodging-houses, and the improved arrangements of vagrant wards, can have been without influence in reducing small-pox. As regards vaccination, the passing of the Compulsory Vaccination Act, in 1853, was followed for ten years by a reduced mortality from small-pox, but the disease was again epidemic in 1863-5. Improved machinery for the enforcement of vaccination was supplied by the successive Acts of 1867 and 1871. The effect of the former of these is masked by the great epidemic of 1871; but since 1871, there has been, in successive quinquennia, a steady diminution until the last, in which there has been a slight rise, concurrently with the growing proportion of children who escape vaccination, and the increase of inefficiently-performed private vaccinations. To the alteration in the age-incidence of small-pox since vaccination came into vogue I need only allude. It is only during comparatively recent years that hospital isolation of small-pox cases has been systematically practised. In earlier years, hospitals were built or extemporised on the occasion of an epidemic, but were rarely kept in readiness for the isolation of the early cases. Even now, their provision is the exception rather than the rule, except in the larger towns. In London, the hospitals of the Metropolitan Asylums' Board have been available for small-pox since 1870; but nevertheless, the mortality from small-pox remained high. It was proved by the investigations of our Jenner Medallist, Mr. Power, that the small-pox hospitals within the confines of the Metropolis formed foci for the dissemination of the disease around them; and in 1886 these hospitals were closed for small-pox, cases of that disease being taken down the river to

hospitals outside the area of London. Since then, the death-rate from small-pox in London (the deaths among cases so removed being included) has undergone a marked diminution, as shown by the following figures :—

	1870-75.	1876-80.	1881-85.	1886-90.	1891-96.
Small-pox death-rate per million living ...	604	310	287	2	16

(see also Chart VI).

The principal agents by which small-pox is spread at the present time are tramps and unrecognised cases. Unfortunately, while vaccination has caused many cases of small-pox to be of a mild and modified form, less readily recognisable than the typical disease, isolation of small-pox has deprived many medical men of the opportunity of becoming familiar with it; and thus it occasionally happens that the nature of a case of small-pox is overlooked until the mischief is done. Outbreaks of small-pox also occasionally occur among sorters of rags at paper mills: the infection of small-pox being especially persistent, and attaching itself readily to textile articles.

Influenza, which, as a registered cause of death after the epidemic of 1847-48, gradually declined almost to extinction, has, since its return in the winter of 1889-90, become one of our most formidable epidemic diseases, having caused in the quinquennium 1892-96 a mortality of 326 per million: greater than that in any preceding quinquennium, and less among epidemic diseases only than the death-tolls from diarrhœa, measles, and whooping-cough; while, unlike these diseases, its victims are, largely, persons of ages when their life is of most immediate value to the community (see Chart V). The mortality from influenza, though considerable every year since its return in 1889, has, however, steadily declined since 1891, with the exception only of 1895, and its behaviour has changed. Instead of the rapid spread, abrupt culmination, and speedy decline to almost complete disappearance which marked the earlier epidemics, the later ones have become more protracted, and the disappearance in the intervals less complete; in fact, influenza has taken on the character of an endemic disease with seasonal increase in the winter months. I fear that we must admit that influenza remains an opprobrium to preventive, if not to curative, medicine, and it seems likely to do so until its pathology is better known. It is, I believe, now generally accepted that the disease is propagated from person to person, and that Pfeiffer's bacillus is its essential cause; but the crux is to determine

what the factor or factors may be, under the influence of which it from time to time assumes such a remarkable increase of virulence—often nearly at the same time in distant and dissimilar parts of the world. In order to carry the investigation of influenza further, the chief requisite appears to be some method, such as we have for enteric fever or diphtheria, of identifying the true disease and discriminating between it and the ordinary catarrhal affections which resemble it, and are often called by the same name.

The last disease which I shall notice is Phthisis. From this disease (formerly so dreaded on account of its supposed incurability and inevitable heredity), the mortality has steadily declined in each succeeding quinquennium, until it is now less than half what it was fifty years ago. In the period 1847-51, the death-rate from phthisis per million living was 2,872, and in 1892-96 it had fallen to 1,405 (see Chart V). Other tubercular and scrofulous diseases have also declined, but the comparison with earlier periods is somewhat affected by changes in classification. As I have already remarked, the pioneers of sanitary reform in the early forties recognised the influence of unsanitary conditions in promoting the endemic prevalence of consumption, and its decline is doubtless largely attributable to the general improvement which has taken place in the conditions of lodgment of the people: such as the closing of cellar dwellings, the repression of overcrowding, the proper provision of ventilation of houses and air space about them, and the ventilation and abatement of dust in factories. The late Sir G. Buchanan also showed that a considerable abatement of phthisis mortality followed works of sewerage and drainage, where these had the result of lowering the level of the subsoil water. Light has been thrown upon the mode in which such circumstances affect the prevalence of phthisis by the observations of Dr. Ransome and others. that the bacillus tuberculosis long retains its vitality in dark, damp, dirty places, but soon perishes if exposed to light and air. Attention is now being paid to the question of direct attempts to prevent the spread of tubercular diseases by disinfection of the sputa of consumptive patients, and by preventing the sale of milk yielded by cows affected with tuberculosis, and of the flesh of tuberculous animals.

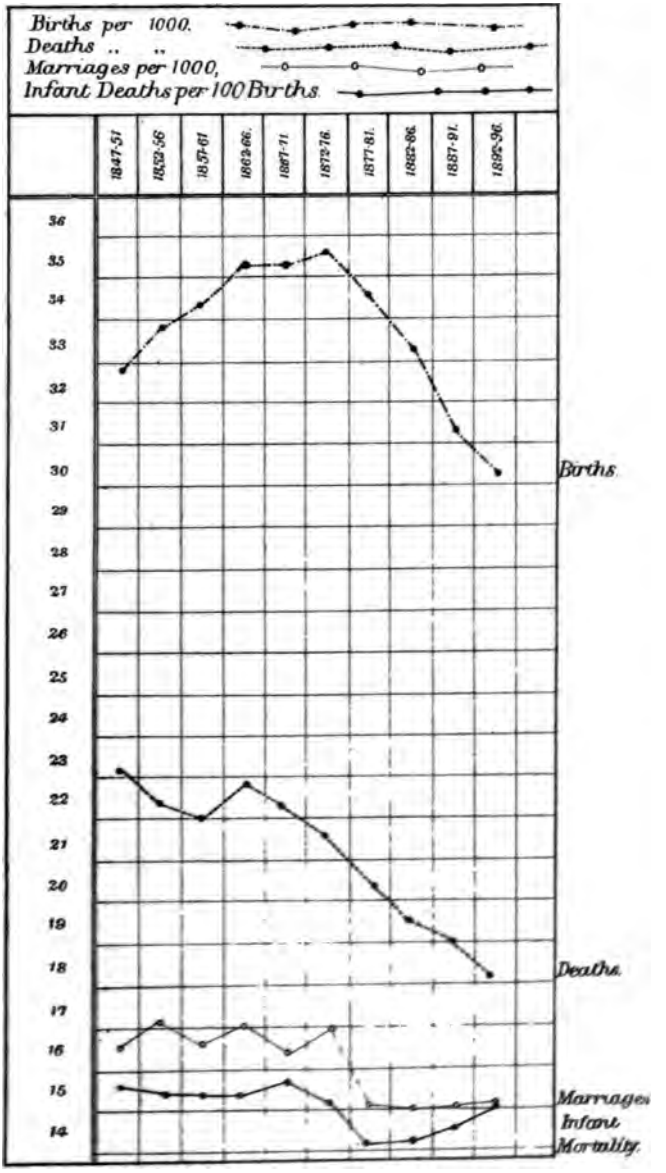
In conclusion, we may observe that for the production of an epidemic of any disease, the concurrence of three factors or groups of factors is necessary. There must be, first, the

presence of the specific contagium; secondly, a favourable environment in the form of appropriate conditions meteorological, topographical, social, or sanitary; and thirdly, personal predisposition on the part of those who are exposed. The environment may act by affording facilities for bringing the contagium into relation with its victims, as a polluted water supply affords opportunity for the spread of cholera and enteric fever; by enhancing the virulence of the contagium, as appears to happen from time to time with influenza; or by lessening the powers of resistance of the persons exposed to it, as famine predisposes a community to typhus.

In our efforts to prevent the spread of epidemic diseases we may consequently aim to counteract one or other of these three factors. We may seek to exclude the contagium, as was formerly attempted by quarantine, to remove it by isolating the patient, or to destroy it by disinfection. The veterinarian has a still more drastic method which we cannot employ. We may seek, by improving the sanitary state of a locality, to remove the conditions which favour the propagation of disease, a method which has borne such good fruit in the case of cholera and enteric fever; or, thirdly, we may seek to render the persons who may be exposed to infection proof against the disease: a method which has been long in use in the instance of inoculation and vaccination against small-pox, and has recently been practised, in one mode or another, against other diseases, as plague, enteric fever, and cholera.

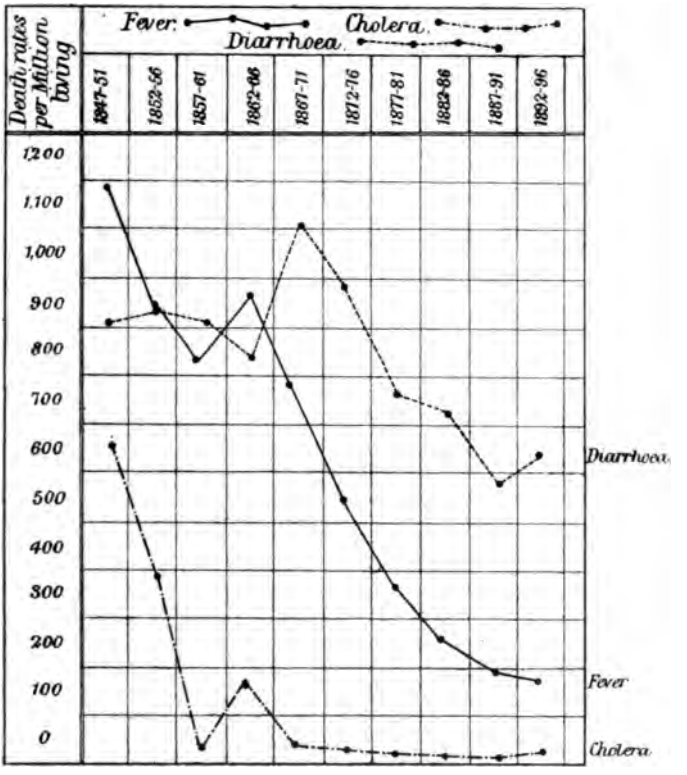
Our efforts for the prevention of epidemic diseases will achieve success in proportion as the natural history and causes of these diseases are known; as the practical means by which these causes can be removed or counteracted have been designed and perfected; and as public opinion has been educated to see the need of making use of these means. The first of these functions, viz., that of elucidating the behaviour and causes of epidemic diseases, is the one which appertains to our Society; and it only remains for me, in thanking you for the patience with which you have listened to this Address, to express the hope that our labours during the session upon which we have entered may be fruitful to this end.

CHART I. ENGLAND AND WALES, 1847-96.



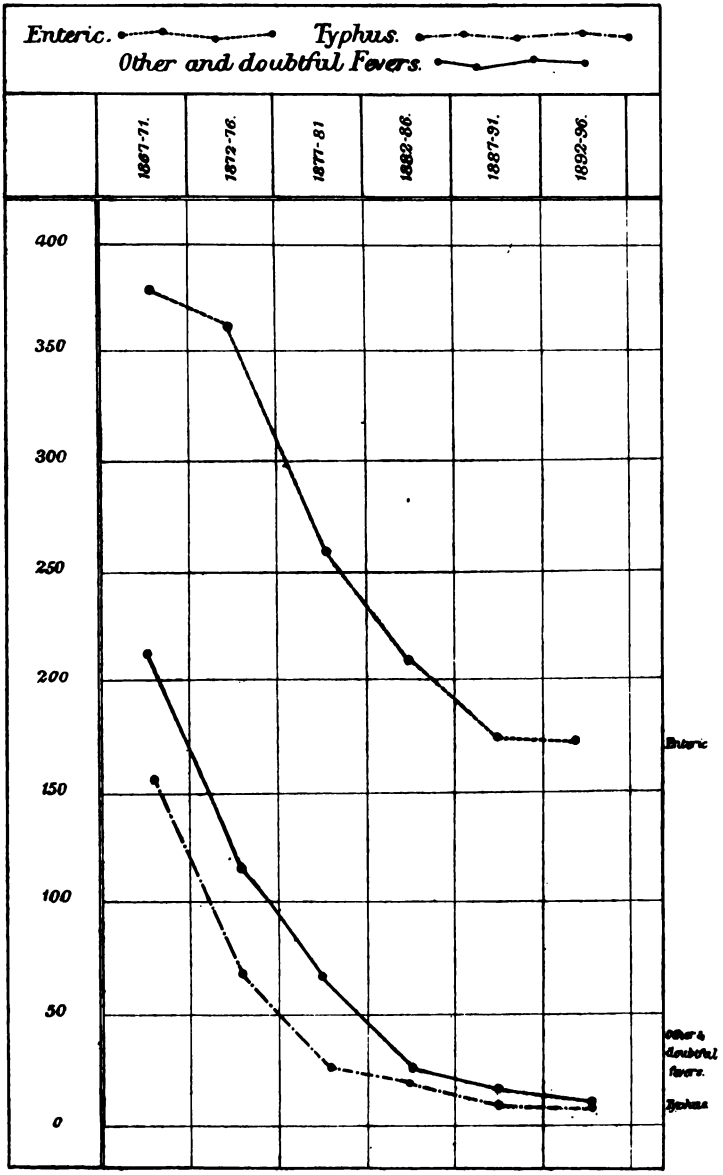
Birth, Death, and Marriage Rates per 1000 Inhabitants and Infant Mortality in Successive Quinquennia.

CHART II. ENGLAND AND WALES, 1847-96.



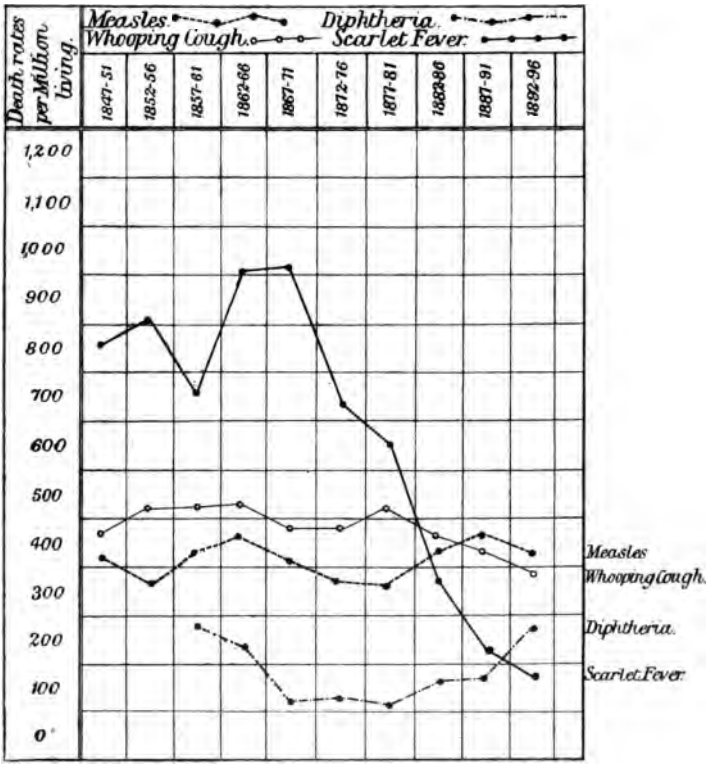
Death Rates per 1,000,000 Inhabitants in Successive Quinquennia from Fever, Cholera, and Diarrhœa.

CHART III. ENGLAND AND WALES, 1867-96.



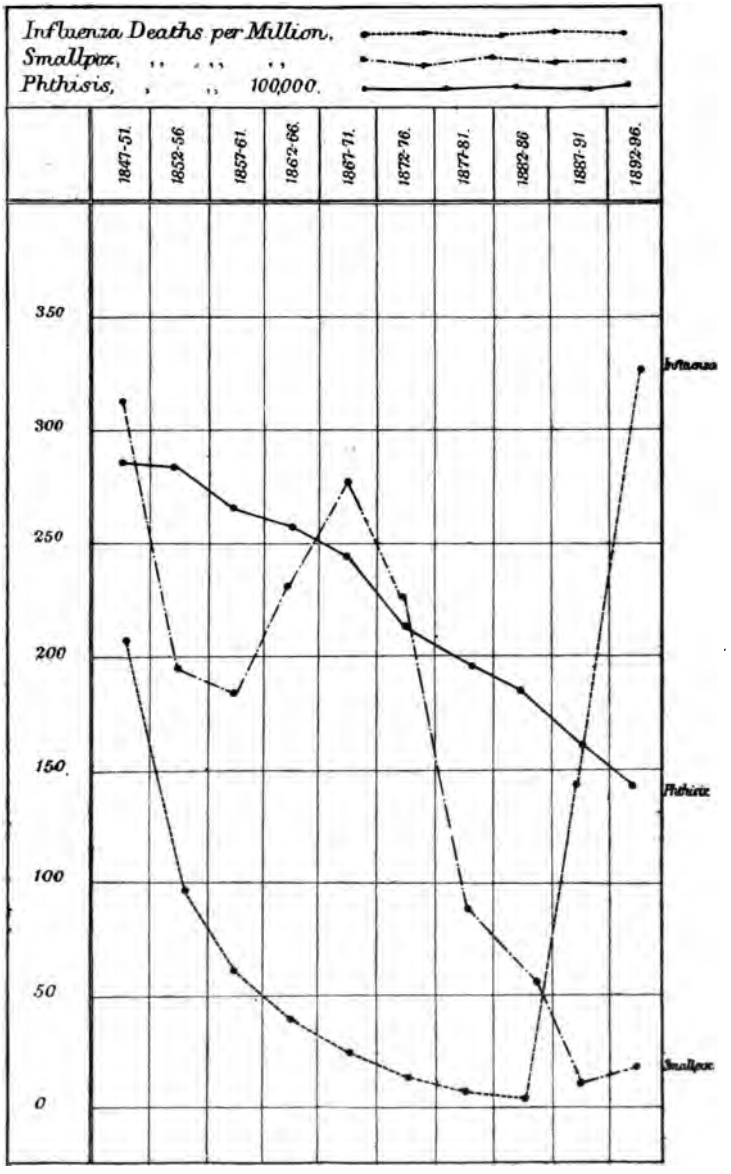
Death Rates per 1,000,000 Inhabitants in Successive Quinquennia from Typhus, Enteric, and Other continued Fevers.

CHART IV. ENGLAND AND WALES, 1847-96.



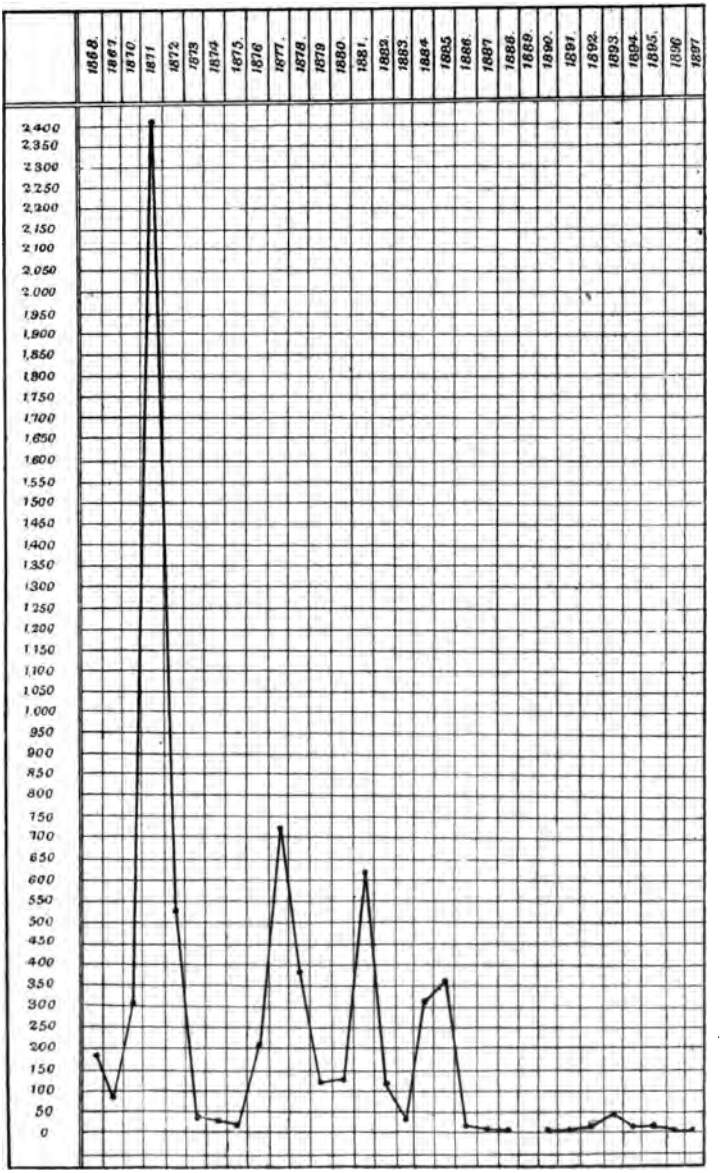
Death Rates in Successive Quinquennia from Diphtheria, Scarlet Fever, Measles and Whooping-Cough.

CHART V. ENGLAND AND WALES, 1847-96.



Death Rates per 1,000,000 Living from Small-pox and Influenza, and Death Rate per 100,000 Living from Phthisis.

Chart VI. London, 1868-94.



Death Rates per 1,000,000 Inhabitants from Small-pox in each Year.

THE PROSPECT OF ABOLISHING TUBERCULOSIS.

By ARTHUR RANSOME, M.D., F.R.S.

(*Read: December 16th*, 1898.)

At the outset of the campaign about to be undertaken by the new Society for the Prevention of Tuberculosis, it is important to all who are interested in the public health that the ground to be covered should be carefully surveyed; that we should reconnoitre the forces opposed to us; that we should know accurately what allies, and what circumstances, are likely to help us, and discover what steps should be taken in order to secure a successful issue. When this has been done, we may perhaps be in a position to calculate the chance of abolishing this terrible disease.

In the present paper I propose to open a discussion on all the points mentioned, and I hope to elicit opinions from the members of this important Society which will greatly forward the object we all have in view, and which will materially assist the operations of the new crusade against tubercle.

A.—The Extent of the Undertaking.

The first part of the work has necessarily to do with figures, and these at present can hardly be said to lead us as far as we could wish. We know fairly well that the disease is almost ubiquitous; that, at any rate, it exists everywhere throughout this country; and that it is especially prevalent where large bodies of people are massed together. But we have to deal with the past as well as the present, and we have no figures of any value that take us back more than about forty years.

The Registrar-General's tables commenced in the year 1838. It is difficult, however, to assign to any of them their right value. There must have been errors in diagnosis in the past, as there are now, and it is not certain that our forefathers made more mistakes than do the present generation of medical practitioners. There was then, as there is now, the tendency on the one hand to exaggerate the nature of the disease which has caused death, and to make it appear that death was inevitable;

and on the other hand there was the reluctance to place a stigma upon a family escutcheon by writing down the name of what was considered to be an hereditary disease. Perhaps these two opposing sources of fallacy may to some extent balance one another, and the final result may approximate to the truth; but in some parts of the country there is another still more serious cause of error, namely, the registration of uncertified deaths by relatives; and in Wales nearly every illness accompanied by wasting is put down by these persons as due to consumption. Yet the figures, as they stand, must be taken for what they are worth, for they are our only means of appreciating the extent of the disease, both now and in the past. Moreover, we may remember the wonderful accuracy of many of the late Sir W. Farr's deductions from statistics which were even more imperfect than these. Fortunately, the figures are of such a nature as to over-ride the possibility that our conclusions could be materially affected by errors in one direction or another. I have already called attention to them, in a comparision between tuberculosis and leprosy (*The Lancet*, July 11th, 1896).

From the accompanying Chart it will be seen that there has been an almost steady decline in the former disease—tuberculosis—in this country, since the date of the first records of the Registrar-General in the year 1838.

In the year 1838 the phthisis-rate stood at the enormous figure of over 3,800 per million of population; but in 1896, the latest year for which as yet we have records, it was only 1,305: about one-third of its former fatality. A straight line drawn from the highest to the lowest point shows how steady and regular has been its decline. If phthisis were to continue to decrease at the same increasing rate of diminution for another thirty years, it would then have entirely disappeared. When the rate of mortality from phthisis is broken up into groups at the different ages, a still more striking picture of its tendency to disappear is given.

It has been possible to make this calculation since the year 1861; and in the last return of the Registrar-General, Dr. Tatham gives a very interesting Table (p. xxvi), which is well worth transcribing.

From this Table (I) it would appear that phthisis has diminished, especially among women, who were at one time the most frequent victims to the disease: "the decrease among males and among females being equal to 40 and 54 per cent. respectively."

living, 1838 - 1894.

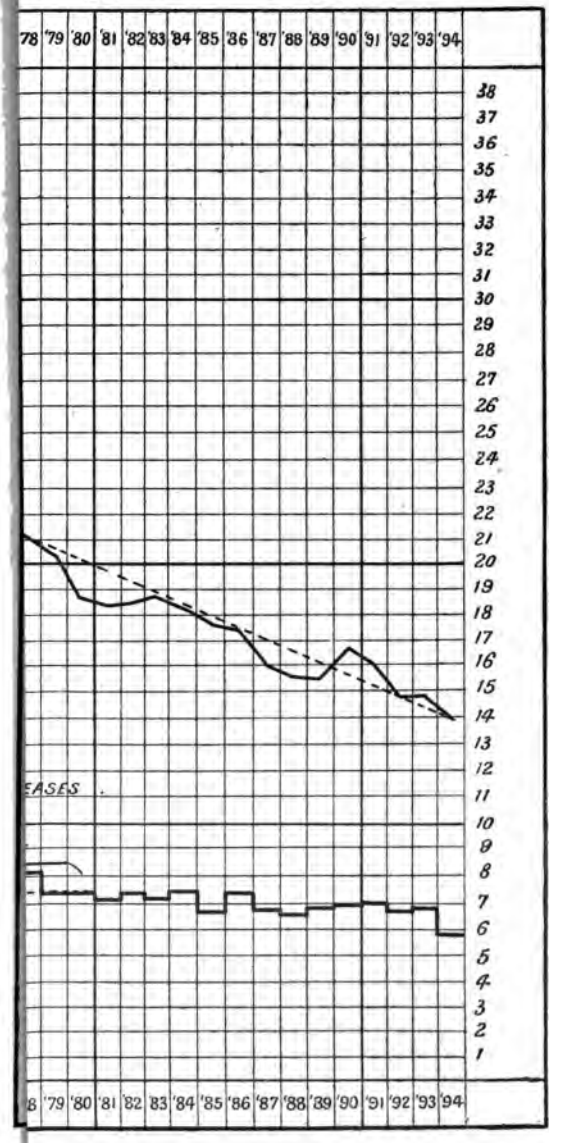


TABLE I.—*Mortality from Phthisis in Groups of Ages*, 1861-96.
(Per Million at each Age.)

	Periods.	All Ages.	0-.	5-.	10-.	15-.	20-.	25-.	35-.	45-.	55-.	65-.	75-.
Males.	1861-70	2467	990	431	605	2190	3883	4094	4116	3861	3297	2024	659
	1871-80	2209	783	310	481	1675	3092	3699	4120	3860	3195	1925	603
	1881-90	1847	553	253	342	1287	2333	3024	3562	3488	2916	1816	688
	1891-95	1633	467	197	260	1076	2026	2548	3268	3205	2687	1572	563
	1896 . .	1485	392	150	203	913	1848	2285	3029	3043	2599	1329	512
Females.	1861-70	2483	947	477	1045	3112	3967	4378	3900	2850	2965	1239	447
	1871-80	2028	750	375	846	2397	3140	3543	3401	2464	1777	1093	407
	1881-90	1609	518	327	699	1800	2315	2787	2730	2053	1512	974	397
	1891-95	1303	421	260	561	1428	1740	2155	2305	1742	1294	800	350
	1896 . .	1138	349	204	454	1183	1562	1874	2090	1543	1170	676	375

When we take the middle period of life only, the diminution which has taken place in the last thirty-five years is still more striking. At the several ages, from 15 to 35, in the decade 1861-70, the rates for males were 2,190, 3,883, and 4,094. In 1896 the corresponding figures were 913, 1,848, and 2,285: only about one-half (49.7 per cent.) of the phthisis-rates thirty years before.

Among females, the figures in 1861-70 at the same ages, were, 3,112, 3,967, and 4,378; whilst in 1896 they were respectively 1,183, 1,562, and 1,874: a still greater diminution of three-fifths in the thirty years (59.6 per cent.).

In Table II, Dr. Tatham has also kindly calculated for me the rates per million at the age-group 15 to 35 years.

TABLE II.—*Mortality from Phthisis, in England and Wales, at the Age-Group* 15-35 *Years.*

	Periods.	Deaths.	Mean Population.	Phthisis Death-Rate per Million.
Males.	1861-70	118,628	3,420,172	3468
	1896	8,939	5,099,363	1753
Females.	1861-70	144,780	3,701,738	3911
	1896	8,745	5,494,377	1592

These figures only confirm the previous calculation.

The saving of life from this destroying agent alone, in the last fifty years, must therefore be enormous.

The conclusion to be drawn from these figures is that, within the period in question, conditions must have been introduced into the lives of the people which are hostile to the bacillus of tubercle, and which are therefore working in our favour.

It would not be difficult to single out some of the favouring circumstances which have conduced to this astonishing reduction in the rate of mortality from phthisis. I think that it will be granted at once that none of the sources of fallacy before-mentioned can account for it. It is most probable that it has been due to the great changes in the conditions of life which have taken place within the present century; and especially to the attention which, during the reign of Queen Victoria, has been paid to questions of public health.

It may be observed on consulting the Chart, that the first great drop in the phthisis-rate took place in the decade 1840-50, about the time that serious attention began to be given to sanitary reforms, and especially to *land-drainage*. It then remained scarcely reduced for about seventeen years; but from 1867 to 1896 the decrease of the disease has been continuous. It is in this period that most of the great sanitary works have been carried out in this country. We can hardly doubt that it is to them that we owe so substantial a diminution of the disease. We must also give some credit to the higher standard of living among the working classes, to the better and more abundant food, the warmer or more suitable clothing, and above all to the improved dwellings, which have now been built in all our towns for the accommodation of artizans and labourers.*

On the other hand, it certainly cannot be ascribed to any special measures of repression against the disease. It has taken place without any attempts at disinfection, or destruction, of the *materies morbi*, and in spite of the increasing aggregation of the population within the areas of our manufacturing towns.

It will be necessary presently to inquire more closely into the several circumstances which enhance or diminish the virulence of the bacillus of tubercle; but it is desirable

* The fact that women have been the chief beneficiaries points to this last-named circumstance as the chief agent in relief, seeing that females are, for the most part, the keepers at home.

first to estimate the extent to which tuberculosis at present prevails.

In order to ascertain this, we have to return to the Registrar-General's figures. In the year 1896 he tells us that there were 40,251 deaths from phthisis, and 19,040 deaths from other tubercular diseases—in all nearly 60,000 deaths from this disorder.

If we accept the estimate of the average duration of phthisis, which has been made by such competent observers as Drs. C. T. Williams, James Pollock, and others, at something over four years, we shall have to conclude that over 160,000 consumptives, and perhaps 30,000 or 40,000 persons affected by other tubercular disorders, are now existing in England and Wales; in all about 200,000 persons who are capable of producing the material of the disease; and, if we include Scotland and Ireland, probably not fewer than 300,000 persons are thus affected within the British Isles at the present time. But even this vast army of sufferers hardly represents the whole force which we have to control, and to prevent from spreading the disease, for there are certainly many thousands of others who are as yet unconscious of its presence, but who are nevertheless capable of doing mischief.

We are told by many eminent pathologists that from 20 to 30 per cent. of all persons dying in hospitals between the ages of twenty-five and seventy-five show signs of healed tubercular lesions. Many of these patients had never been made aware of their condition; and there must be a multitude of others in whom the disease has not yet been detected, who are only waiting to join the ranks of those who are already enrolled, and who are even now as dangerous as our more open enemies.

Last, and probably not least, if we are to take account of all possible sources of tuberculous virus, we must not forget the hundreds of thousands of cattle, and other animals, which are affected with tubercular disease, which are daily spreading it about broadcast, and which are even less under control in this respect than human beings. It has been pointed out also that most of the animals in our zoological gardens are tuberculous; and, although avian tubercle is supposed to be incapable of infecting the higher order of animals, Prof. Nocard, of Alfort, has recently shown that, under certain conditions, the virus is capable of modification, so as to become a source of danger.

B.—The Sources of the Strength and Weakness of the Bacillus.

In these days of bacteriological research there is nothing wonderful in the fact, that so small a cause as a tiny vegetable mould can produce such a vast amount of evil. It is one of the "weak things of the world" which the Almighty has chosen "to confound the things which are mighty;" and we may perhaps be allowed to surmise that their action is permitted, in order to bring men to a knowledge of His beneficent "Laws of Health." At all events, their activity has had this result; and in any case we are bound to search for the sources of its great power for evil. These are:—

1. *The immense number of the enemy.*—Tuberculous patients are to be found in all communities of men, and the amount of virulent material which is dispersed by them is probably enormous. It has been calculated that about two millions of bacilli are often contained in a single portion of sputum, expelled from the lungs of a tuberculous person.

2. *The tenacity of life of the bacillus under certain favourable conditions.*—I have myself found that it retains its virulence for three months, when kept in a small insanitary cottage in Ancoats; and other observers have recorded much longer periods of infectiveness. Under these unhealthy conditions, tuberculous sputum has been found to resist all the changes of the seasons: wetness, dryness, freezing, considerable heat, snow, rain and hail.

I have also found that the bacillus will grow upon pure filter paper, if kept saturated with organically-charged fluids, condensed from the vapour of ground air, from healthy and diseased breath, from humidified weaving sheds, and from cellars.

3. *The contamination of milk by tuberculous material.*—This is another important source of infection, and meat and other kinds of food are probably sometimes media for conveying the disease.

4. *The chronic character of the complaint.*—This is itself a powerful influence in its favour. It prevents the early recognition of the disease; it permits a dangerous kind of intercourse with other individuals: especially when the affected person indulges in the filthy habit of expectorating in an unhealthy dwelling, or in an ill-ventilated workshop; thus facilitating the constant and long-continued manu-

facture of tuberculous dust, and so endangering the lives of his companions and fellow-workpeople.

5. *The long incubation period of the disease.*—This often prevents the source of infection from being recognised, and, its origin being thus concealed, measures for preventing its spread to others cannot be undertaken until it is too late.

6. *The habit of the specific microbe of contracting alliances* with other organisms, such as the tetragonus, streptococci and staphylo-cocci. This habit enhances its power for evil, both by increasing the fatality of the disease, and by preparing the way for its entrance into the human body.

7. *The power possessed by the bacillus of producing "toxines."*—This probably helps somewhat in the same direction as the habit just mentioned.

8. *The readiness with which the complaint can be transferred to cattle,* and to other domesticated animals. This increases the scope of its action.

9. *Lastly, the failure of ordinary disinfectants to kill the microbe.*—This increases the difficulty of dealing with it.

It will be seen, therefore, that small though it be, we have no insignificant foe to meet; yet, on the other hand, there is no need to despair of successfully coping with it.

In the great discussion on tuberculosis, in the year 1885 (*British Medical Journal,* 1885, vol. i, p. 130), Dr. Moxon made the striking remark that "The life of the bacillar parasite is difficult, easily discouraged by unfavourable circumstance, like an aphis by an easterly wind;" and I think we shall find that his statement is strictly in accordance with the facts.

There are many conditions adverse to the bacillus of tubercle; otherwise we may be sure that, without special measures of repression, it would hardly have given ground, as we have seen that it has done. Some of them may be briefly considered:—

1. *The bacillus has a constant tendency to lose virulence,* and even to decline in the vigour of its growth. Under circumstances most favourable to its longevity, it usually diminishes in power; and, unless it is passed through the bodies of animals, it cannot be made to retain its virulence. I am informed that most of the specimens which are to be found in our bacteriological laboratories are non-virulent. The successive cultures gradually lose their strength; and even simple keeping, under favourable circumstances, will not preserve its properties.

2. *It is extremely susceptible to the action of sunlight*

and air, in spite of its resistance to certain of the meteorological influences which have been already mentioned.

In a research devoted to this subject by Prof. Delépine and myself, in one series of trials, all the experiments (*Researches on Tuberculosis*, the Weber-Parkes Prize Essay for 1897, Smith, Elder, and Co., pp. 21-22) showed that very short exposures to sunlight and air sufficed to deprive the organism, even in sputum, of all virulence. In one case (Table XIII, No. 190), a specimen exposed to light and air for two days only, with but little radiant sunshine, was found to have entirely lost its power for evil. Another specimen, exposed for three days, with only one hour of sunshine, gave a similar result (Table XIII, No. 156).

Ventilation alone, in the dark, also diminishes, though it does not necessarily destroy, virulence (*loc. cit.*, pp. 18-19).

It is important to notice that, in all the experiments on light and air, the times of exposure ascertained to be sufficient to disinfect the temporary habitat of the bacillus, were less than would suffice for the pulverisation of the sputum, under ordinary circumstances; yet specimens of the same sputum gave tubercle to guinea-pigs, after it had been kept in the dark, with little air, for thirty days. *In other words, virulent tuberculous dust cannot be formed in well-lighted, well-ventilated places.*

Sunlight and air are probably the most powerful disinfectants known of the material of tuberculosis.

3. *Fortunately, man is one of the animals refractory to the action of the bacillus.*—In man, when infection takes place through the skin, the poison is usually arrested at the first lymphatic gland, and is then often discharged from the body by suppuration. When the virus makes its entrance by the respiratory tract, it is highly probable that it cannot take root, unless some previous injury has afforded it a resting-place, or some abrasion has given it an entrance into the system.

4. *The long period of incubation*, though it disguises the source of infection, is yet an important safeguard to the individual. No infection from tuberculosis can take place unless the bodily tissues are in such a condition as to receive, and to nourish into colonies, the bacilli which have found an entrance. This condition is usually the result of some state of the bodily constitution, due to some predisposing injury, or some weakness, hereditary or acquired.

In a perfectly healthy human body, there are a multitude of barriers against the establishment of colonies of the bacillus, barriers which are hardly ever entirely overcome.

Amongst the more important of these defences may be mentioned: 1. the aqueous vapour of the breath, which entangles all solid particles in its meshes and deposits them in the glairy mucus lining of the air-passages; 2. The continual working of this mucus, by the currents of air, and by the action of the "cilia" lining the air channels towards the mouth; 3. The presence of protecting glands extending from the throat to distant ramifications of the bronchial tubes; 4. The action of aggressive warrior elements, the "phagocyte" cells.

The so-called "giant cells" have apparently this function; and it is tolerably certain that there are other cells in the lymphatic glands, and in other parts, whose office it is to seek for and to devour intruding bacilli and other organisms.

5. *Koch's discovery of "tuberculin."*—As a test for the presence of tubercle, this will certainly in the future be of enormous service in assisting us to stamp out this disease in cattle.

6. *The drying of the subsoil.*— The influence of this upon the prevalence of phthisis is as yet imperfectly understood; but no doubt can now be entertained that thorough drainage and drying of the soil are important agents in preventing the disease.

There are many other influences—some hostile, others predisposing, to tuberculosis. Among these we may place hereditary weaknesses of certain organs of the body, especially of the lungs, which produce susceptibility to contract the disease, and increase the tendency to a rapid and fatal issue. It is well known also that there are certain disorders which leave behind them similar proclivities; such complaints, for instance, as enteric fever, measles, and others, which are likely to end in permanent disablement of portions of lung tissue. Other affections, tending to general debility, are likewise favourable to tuberculosis, such as diabetes, syphilis, alcoholism, cancer; and pregnancy seems in some persons to have the same effect. Susceptibility to tubercle is often acquired by persons who have suffered from other diseases of the lungs and pleura; or from external injuries affecting the joints, the skin, or other parts of the body.

Broncho-pneumonia and cirrhosis of the lungs, arising from inhaling irritating dusts, are peculiarly likely to lead to the further deposit of tubercular material; as the bacilli often inspired along with the dust, find suitable resting-places within the damaged tissues.

When we add to these conditions, so favourable to the enemy, the existence of overcrowded, unwholesome dwellings, of workshops insufficiently supplied with air and light, of theatres and other places of public assembly badly ventilated, and seldom visited by rays of sunlight, we shall not have much difficulty in accounting for the differences in rates of mortality from consumption and other forms of tubercular disease.

There are whole areas of insanitary houses, in most of our large towns, where the disease prevails three or four times as much as in the more healthy districts; and it is sadly interesting to compare the incidence of phthisis upon the classes of workpeople employed in trades where the ventilation of workshops is not compulsory, with the relative immunity of those to whom plenty of air is supplied.

C.—The Prospect of Victory.

This brief review of the opposing forces may perhaps enable us to form some idea of the way in which the campaign now opening will end. In the first place, it is of happy augury for the result of the contest that our enemy is at present in full retreat. It is well known that a campaign which begins with victory is often carried out by the victor with great advantage. The common phrase that "nothing succeeds like success" tritely expresses this fact. But, in the present case, the dispersal of a large contingent of the army of tubercular disease should do something more than animate us for the final contest. It points the way to further victory.

We have already seen, that the great and steady decline in the phthisis-rate coincides in point of time with the introduction of measures of sanitary reform, and with the improvement in the general conditions of life which have taken place during the reign of Queen Victoria: it cannot be traced to any special measures of repression, such as isolation of phthisical persons, or disinfection of sputum, or of houses. Surely it should encourage us to put in the forefront of the coming battle all the means which have already proved so successful.

(*a*) It is imperative, therefore, that all sanitary authorities should put in force the manifold powers which they now possess for improving the public health; not only that they should attend to the sewerage, and house and subsoil drainage, but that they should turn their attention to the still more important question of removing from their streets and dwellings, and places of social assembly, the more subtle

and even more dangerous "air sewage." Owing to the prevalence of the disease among cattle also, similar measures will have to be taken with regard to cow-sheds, etc.

Viewed in relation to our present subject, the prevention of phthisis, the problem of removing organic impurities from the air we breathe is a very wide and difficult one. It involves not only the admission, without draught, of a certain number of cubic feet of air to living rooms or to places where human beings congregate, but that the air so admitted should be itself fairly pure.

It is important, doubtless, that all insanitary property should either be destroyed or made fit for healthy habitation; and that back-to-back houses, undrained and unaerated basement dwellings, should be done away with; that workshops and factories should be efficiently ventilated; that schools and places of public assembly should be more fully supplied with a flow of air adequate to the needs of their temporary inmates. But all this will be of comparatively little use if the outer air be in such a condition as to induce all cleanly people to close their windows against it.

The outer air must therefore be kept as pure as possible; in the streets, free course must be given to the wind; there must be no blind alleys or streets closed at one end; and, wherever possible, the width of streets must be proportional to the height of buildings, and an adequate space must be left around each building. By this means both sufficient air and sufficient light may have access to the dwellings, and the air will be freed from much of its impurity.

Local authorities will, therefore, have to put in force their strongest powers for the prevention of pollution of the air, by smoke and noxious vapours; they must provide ample lung-space in the shape of public parks and open playgrounds; and they will have to carry out extensive works of reconstruction of insanitary areas.

Building bye-laws must be carefully scrutinised, and if necessary amended, and the various "Rookeries" Acts must be ruthlessly put in force. When all this is done, we cannot doubt that the great shrinkage in the mass of tubercular disease will continue to go on. I am the more anxious to emphasise this point, because it seems to me that most of the anti-tubercle societies are inclined to neglect this vital subject, for disinfection and other matters of somewhat less importance.

(*b*) In the next place, if, by means of disinfection of sputum and destruction of tuberculous dust in dwellings,

and places of public assembly, we cut off the supplies of infectious material, the assault of the enemy must fail for want of ammunition. Let it be clearly understood, however, what is involved in this method of carrying on the war. It will be necessary to bring into action all the time-honoured machinery of notification, disinfection, isolation (in certain cases), and hospital accommodation.

(*c*) *Notification.*—It is important that the full extent of the attacking force should be known. There is an "intelligence department" in every army.

It is essential, therefore, that every case of tuberculosis, as soon as its nature and locality are known, should be notified to the Medical Officers of Health in every district. It does not follow that any immediate action should be taken by him. We are told by Dr. Hermann Biggs that, in New York, cases in private houses, and under the care of private physicians, are not in any way interfered with. "The data regarding such cases are recorded, but they are not visited by the inspectors of the Department; no circulars of information are sent to them, nor are they interfered with in any way, except by special request of the attending physician. The Department has always fully recognised the importance of non-intervention between the physician and his patient in tuberculosis, in cases in which the intelligent practitioner may be trusted to give the simple instructions necessary to prevent the transmission of the disease to others."

In that city, however, "cases of tuberculosis, reported to the Department by the public institutions of the city, are visited at their houses by medical inspectors, specially detailed for the purpose, who give verbal directions respecting the proper care of the sputum, and leave circulars of direction. Cases requiring hospital treatment are visited by a special inspector, who examines the patient, and collects some of his sputum, which is afterwards examined in the laboratories of the Department."

If tubercle bacilli are found, and the patient is undoubtedly tuberculous, and is not in circumstances to receive proper care and medical attendance, or is a source of danger to others, and desires hospital treatment, he or she is removed, by order of the Department, to one of two specially designated hospitals.

I have described the New York method of notification and its sequences thus at length, because it seems to be worthy of careful consideration. It affords intelligence of all overt cases of the disease, and yet is followed by action

only in those cases where such action seems needful for the protection of the rest of the community.

(*d*) *Disinfection*, either under the guidance of the regular medical attendant, or of the Medical Officer of Health, is the natural sequence of notification. There are published many admirable leaflets of directions how to deal with tuberculous material: these will have to be distributed to every house inhabited by a consumptive or tuberculous patient. The dwellings of such persons will have to be thoroughly disinfected by Professor Delépine's methods, sometimes during the life of the patient, always after the removal to other quarters, and after death.

(*e*) *Isolation* may be necessary in the case of unteachable persons, or where there is such destitution that the proper precautions cannot be carried out. For this purpose—

(*f*) *Hospital accommodation* will be required. At the present time, in most parts of England, such accommodation is only afforded by the workhouse authorities; but I would submit that it would be a legitimate expenditure on the part of local boards of health if they were to provide male and female wards for the reception of such cases, in connection with their hospitals for infectious diseases.

(*g*) There is yet another very important weapon in our armoury for the abatement for tubercular disease, and that is the *abolition of the complaint* in our herds of cattle. It is doubtless true that if all milk were boiled, and all meat thoroughly well cooked, there would be little danger of contracting disease by the medium of the articles of food; but this precaution would not obviate the risk of infection from the tuberculous material scattered about the byres by the cattle themselves. Moreover, who is to make sure that the precaution is always carried out? There will always be careless people who will not take the necessary trouble, and there are many children who will not take boiled milk.

The only way of avoiding all these dangers is to form herds of tubercle-free cows. As Bang, in Denmark, has shown, it is quite possible, without much expense, to extinguish the disease within the generation of cattle which it affects: "provided sufficient means are taken to protect the succeeding generation from becoming affected."

The same object has been attained more rapidly in England, notably by Lord Vernon, of Sudbury. It needs, therefore, only a strong effort so to influence public opinion, that the use of tuberculin, as a test, should become general

throughout the country, and that all herds of cattle should be rendered innocuous to the public health.

It is evident from the foregoing facts, in the first place, that the forces opposed to us are steadily on the decline; and that this diminution in the disease must be attributed to the inherent weakness of the microbe, as compared to the powerful influence of ordinary sanitary reforms, without special measures of repression; and, in the second place, that we possess in disinfection, in the tuberculin test, etc., potent weapons of precision, with which, when fully put into action, we may fairly hope to accomplish the final conquest of the foe.

The question naturally arises, When may we expect complete success? When will tuberculosis cease out of the land?

Judging from the past, I think that we may look for this consummation before very long, and without waiting for a Utopian state of things. Already the rate of diminution in the prevalence of phthisis is greater than that of leprosy, when that disease began to decline. It took at least two hundred years for leprosy to diminish to any appreciable extent; but sixty years has marked a decline in phthisis of at least two-thirds. When all our forces are brought fully into action, we may surely expect that at least as great a rate of decline will be continued; and, in this case, another thirty years should see its vanishing point.

We can hardly doubt that the retreat will go on at an increasing speed. When all health authorities perform efficiently the several tasks which have now been set before them; when, by the means which have been mentioned, tuberculous cattle have been abolished from our herds; when the general public have been fully instructed how to deal with tuberculous material; and when, by means of "open-air sanatoria" in connection with all our great towns, the existing consumptives, and other tuberculous patients, are provided with the requisite essentials for their cure, then may we hope for complete success.

Let us take, then, for our motto, "Victoria, et per Victoriam, Vita."

EPIDEMIC CEREBROSPINAL MENINGITIS.

By R. BRUCE LOW, M.D.

(*Read: January 20th*, 1899.)

EPIDEMIC cerebrospinal meningitis does not, at first sight, seem a subject that is likely to excite much interest in the minds of English epidemiologists, for the reason that the disease is not regarded as one of frequent occurrence in this country. Papers describing certain outbreaks of epidemic cerebrospinal meningitis have, however, already been read before this Society on several occasions. In July, 1865, a paper by Professor Hirsch, of Berlin, was read, giving an account of the severe epidemic of the disease which had appeared in that year in North-East Prussia; (1)* and in December of the same year Dr. Brown, of Rochester, (2) gave details of some groups of cases that had come under his notice. In July, 1867, Professor Mapother, of Dublin, (3) read a paper "On the Malignant Purple Fever Epidemic in Ireland," cases of which had come under his observation in Dublin in 1866 and 1867: and at the same meeting, Staff-Surgeon Marston (4) furnished an account of what he termed the epidemic of "malignant purpuric fever in Ireland, as it occurred among the troops" in 1866-67. In March, 1888, Dr. Bolton Corney contributed a paper on "Epidemic Cerebrospinal Fever in the Fiji Islands (5) in 1885." Since 1888 no papers on this disease have been read before the Society; but in the meantime considerable additions have been made to the common fund of knowledge concerning epidemic cerebrospinal meningitis. It appears to me, therefore, that the present is an appropriate time for taking stock, as it were, of the facts that have been gathered during the last ten years concerning this disease, which has long been regarded as a mysterious malady.

From facts that have come under my own notice, I am led to think that the characteristic features of epidemic cerebrospinal meningitis are but little familiar to many English medical practitioners. Some who have studied the subject say that in England, as elsewhere, the malady is frequently overlooked, and that deaths from this cause

* These numbers refer to the bibliography at the end of the paper.

are certified under other names. It is not denied that the diagnosis of the disease is at times attended with considerable difficulty. This difficulty is great, for example, in those cases where the malady kills quickly—as it sometimes does in a few hours—before any train of symptoms has been developed, sufficient to enable an opinion to be formed as to the nature of the illness. The disease has no constant or characteristic eruption, by which it can readily be recognised, like small-pox or scarlet fever, though rashes of various kinds are sometimes seen in different outbreaks, while in others no skin eruption at all is visible. Added to this is the unequal development of marked symptoms in cases comprised in the same epidemic; in some cases, as has been said, the malady proving fatal in a few hours, whereas in others only a transient illness is seen, characterised by headache and some feverishness, which quickly passes off. So that in the same epidemic it is not uncommon to meet with abortive, irregular, ill-defined, or anomalous cases side by side with cases of extreme severity. The milder cases are diagnosed sometimes as influenza; or, if lasting some time, as typhoid fever, or tubercular meningitis: the severer cases which prove more quickly fatal are regarded as typhus fever, idiopathic tetanus, or as malignant measles.

It may be useful here to quote shortly a description of the disease:—

The late Mr. Netten Radcliffe (6) defined epidemic cerebrospinal meningitis as "an acute epidemic disease, characterised by profound disturbance of the cerebral nervous system, indicated at the onset chiefly by shivering, intense headache or vertigo, or both, and persistent vomiting: subsequently by delirium, often violent, alternating with somnolence or a state of apathy or stupor: an acutely painful condition, with spasm—sometimes tetanoid—of certain groups of muscles, especially the posterior muscles of the neck, occasioning retraction of the head, and an increased sensitiveness of the surface of the body. Throughout the disease there is marked depression of the vital powers, not unfrequently collapse; and in its course an eruption of vesicles, petechiae, or purpuric spots, or mottling of the skin is apt to occur. If the disease tend to recovery, the symptoms gradually subside without any critical phenomena, and convalescence is protracted: if to a fatal termination, death is almost invariably preceded by coma. After death the enveloping membranes of the brain and spinal cord are found in a morbid state, of which the most

notable signs are engorgement of the blood-vessels, usually excessive, and an effusion of sero-purulent matter into the meshes of the pia mater and beneath the arachnoid."

In the *Nomenclature of Diseases*, (7) issued by the Royal College of Physicians in 1869, the disease was defined as "a malignant epidemic fever, attended by painful contraction of the muscles of the neck and retraction of the head. In certain epidemics it is frequently accompanied by a profuse purpuric eruption, and occasionally by secondary effusions into certain joints. Lesions of the brain and spinal cord are found on dissection."

The above definitions, as will be seen, only cover the severer forms of the disease. It has, however, to be borne in mind that it is quite possible to have epidemic cerebrospinal meningitis without any retraction of the head, or without any eruption of any kind whatever.

Most of those who have written about this disease classify its various forms as follows:—

1. The simple form, having typical nervous symptoms;
2. The fulminant form, attacking suddenly and killing quickly;
3. The purpuric form, attended by hemorrhages;
4. The abortive form, with anomalous symptoms, running a short or irregular course.

In different epidemics these forms tend to run into one another. The mortality varies: in some outbreaks 80 per cent. of the cases have died; in others only 20 per cent. have proved fatal. Those who recover are sometimes paralysed in their limbs or elsewhere: deafness is a common sequela, and in this latter event, when the patient is an infant, it becomes a deaf mute. In America it is asserted that 20 per cent. of the deaf mutes of that country have been caused by epidemic cerebrospinal meningitis.

From the varying character of the malady in different epidemics, or from other causes, the nomenclature of the disease has been very diverse. For this reason there is some difficulty in tracing back in past years the course of epidemics of cerebrospinal meningitis in one or another country, or in collecting statistics relating to the disease, even in the United Kingdom. For example: in some epidemics where meningeal symptoms have chiefly attracted the notice of observers, the disease has been spoken of simply as *epidemic meningitis, epidemic brain fever, epidemic encephalitis*, or, where the victims have mainly been children, it has been at times called *epidemic hydrocephalus*.

In other outbreaks where spinal symptoms have attracted more attention, it has received the name of *spinal fever, cerebrospinal fever, cerebrospinal arachnitis,* or *epidemic meningo-myelitis.*

For a long time this disease was regarded as a form of typhus fever, and certain outbreaks were described as epidemics of *cerebral typhus, cerebrospinal typhus* — or occasionally, when fulminant cases predominated, and where a condition of collapse existed from the first onset, it received the name of *sinking typhus.* In this connection it may be remarked that some of the older epidemics designated variously as *jail fever, spotted fever, petechial fever,* or *hospital fever,* have in all probability been outbreaks of epidemic cerebrospinal meningitis. The details given of the symptoms, together with the accounts of the post-mortem appearances observed in the fatal cases, leave little doubt as to what has been the real nature of the malady.

Some of the most serious epidemics met with, especially in America, have been attended with a purpuric eruption which has varied from small petechiæ to large purpuric patches. , Such outbreaks have in consequence been designated *purpuric fever, nervo-purpuric fever, purpura maligna, infectious purpura, purpura contagiosa, purple fever,* or *malignant purple fever* In this connection it may be mentioned that down to the end of the seventeenth century, a heading under which many deaths appeared in the old London bills of mortality was "spotted fever and purples." In 1686, for instance, 4,185 deaths were returned from "ague and feaver," 314 from "spotted fever and purples," and 66 from scurvy. In 1749 certain deaths were grouped in these bills of mortality as follows: "Ague" 15, "malignant fever and purples" 4,458, and scurvy 5. The so-called "spotted fever and purples," or "malignant fever and purples," has been held by some authorities to indicate merely cases of fever occurring in persons of a scorbutic habit. The experience, however, of the Irish epidemic of 1866-67, which will be referred to further on, strongly suggests that the cases of "purples" have been related more to epidemic cerebrospinal meningitis than to scurvy. The occurrence of large purpuric patches on the skin of persons attacked, during the Irish epidemic just alluded to, caused the disease to receive the somewhat startling appellation of *the black death;* others called it the *black sickness,* the *black fever,* or *febris nigra.* The calling of the disease by the name of "the black death" greatly

accentuated the alarm experienced by the ignorant and superstitious inhabitants of certain parts of Ireland during 1866 and 1867. As is well known, the term "black death" is generally applied to a virulent form of bubonic plague, and its application to any other disease is misleading, and ought therefore to be avoided.

In some epidemics no purpuric spots or patches have been noticed, but in other outbreaks a considerable number of cases have shown crops of herpes upon the face and lips, or elsewhere. This was noticed, for example, in the great epidemic that occurred in 1865 in the Valley of the Vistula, an account of which was given to this Society by Prof. Hirsch, as has already been mentioned. From the occurrence of herpes in a large number of marked cases in such outbreaks, some observers have spoken of the disease as *herpetic fever.*

Owing to the fact that in some large epidemics purpuric eruptions have been present in most of the cases, while in other outbreaks only crops of herpes have been observed, some authorities have inclined to the view that two diseases were in question. But there is now a general agreement among most writers that these differences represent only varieties of one and the same disease. In past years the purpuric variety was seen most commonly in America, while the herpetic type was most frequently observed in epidemics on the Continent of Europe. But this difference is not now so marked as formerly.

One of the most prominent features of epidemic cerebrospinal meningitis is the pain and stiffness in the neck, leading in many cases to retraction of the head. From this, the disease has received in some countries the name of *the stiff-neck fever*, or the *epidemic neck cramp.*

Most authorities are now agreed that the best appellation for the malady is *Cerebrospinal Fever.*

Before proceeding to speak of the distribution and etiology of cerebrospinal fever, it is right to mention that there is a form of cerebrospinal meningitis which, though very closely resembling clinically the specific cerebrospinal fever, is not etiologically related to it. This form appears as a sequela or complication of other infectious fevers, the organisms of such diseases occasionally finding their way to the meninges, and causing a purulent inflammation of the membranes of the brain and spinal cord. Such cases are perhaps not so very uncommon; medical literature furnishes examples of them where cerebrospinal meningitis has developed from an attack of measles, scarlatina,

small-pox, diphtheria, enteric fever, influenza, whooping-cough, croupous pneumonia, tonsillitis, or of gonorrhœa. This form of cerebrospinal meningitis may be also met with in patients suffering from chronic ear disease, or may arise from injuries to the bones of the skull. (8) In separating such cases of cerebrospinal meningitis from cerebrospinal fever, the presence of any signs of other specific infections should be noted, as also the existence or non-existence of ear disease, or evidence of cranial injuries.

From a consideration of the varied nomenclature of the disease, and the difficulties in diagnosis that sometimes occur in epidemics where various forms of the malady appear side by side, and in view of the fact that cerebrospinal meningitis is found as a complication of other infections or of injuries to the head, it is evident that the compilation of any reliable statistics relating to the occurrence of epidemics of cerebrospinal fever must be attended with difficulty, and may prove untrustworthy. I shall, therefore, in what follows, confine myself to a very brief sketch of the distribution of the disease in one and another country, since the recognition of cerebrospinal fever as a separate and distinct disease. I wish here also to point out that the history of the incidence of these epidemics has to be gathered largely from published accounts in medical works and current journals. It is hardly necessary to remind you that epidemics occur in localities where the busy practitioners have little time for writing about them. In the old days, before medical officers of health made their reports on outbreaks in their districts, epidemics often escaped general notice, unless a more than usually devoted practitioner published his experience. Therefore, the absence of records of epidemics of cerebrospinal fever in one country or in another does not imply necessarily that such epidemics did not occur, but that, if they did occur, they were not recorded.

The disease was first described as a distinct specific fever by Vieusseaux of Geneva, who studied an epidemic that occurred there during the winter and spring of 1804-5. Here the onset of the disease was observed to be sudden: it was attended with vomiting, violent pains in the head and neck, and down the spine. In the fatal cases the course was extremely rapid, lasting only twelve hours in some cases, in others from one to five days. In children there were convulsions. In those who died within twenty-four hours of the onset, the larger proportion had an eruption of violet spots upon the body.

Post-mortem examination showed a gelatinous exudation covering the surface of the brain, and a yellow purulent matter on its posterior aspect, as well as upon the inferior surface of the cerebellum and medulla oblongata. Previous to the epidemic observed by Vieusseaux at Geneva, in 1804-5, there had doubtless been other outbreaks of the same nature elsewhere. Since 1805, when the attention of the medical profession was first directed to epidemic cerebrospinal meningitis, many epidemics have been recorded in America, Europe, Asia, and Africa. It has been met with in Iceland as well as in India, in Egypt as well as in England; in hot and in cold countries; in the white as well as in the coloured races.

America.—After the Geneva epidemic the next recorded outbreak was in America, in the state of Massachusetts, in 1806. Since that date, up to the present day, the United States of America have hardly ever been free from the disease. In 1808-9 cerebrospinal fever was epidemic in Virginia, Kentucky, and Ohio; and in 1814-16 in various parts of New England. In 1823 it was in Connecticut, and in 1828 it again appeared in Ohio. From 1842 to 1850 a number of scattered and isolated outbreaks occurred in various parts of the States. During the Civil War, from 1861 to 1864, the malady became widely spread, attacking the troops, specially in the army of the Potomac, and in camp near Washington. It was severely felt among the negroes sent by the Confederates to Memphis. Massachusetts again suffered in 1864 and 1865. In 1872 a considerable outbreak took place in New York. As many as 782 deaths were certified during that year from epidemic cerebrospinal meningitis: the number certified from the same cause in the previous year was only 48. From the official report issued by the State Board of Health, it appears that the first fatal case of any affection at all similar had been recorded in 1861, when "spotted fever" was assigned as a cause of death in a single case. No others were found up to 1866, when 18 deaths from cerebrospinal meningitis were certified. The subjoined Table shows the number of deaths from this cause in New York from 1866 to 1873:—

Deaths from Cerebrospinal Meningitis in New York—Population (1870) 942, 292—*during the eight years*, 1866 *to* 1873.

1866	1867	1868	1869	1870	1871	1872	1873
18	32	34	42	32	48	782*	290

* 492 of these deaths occurred during the first quarter of 1872.

In 1873, a serious outbreak again occurred in Massachusetts, 517 deaths being referred to this cause during the year. Localised outbreaks in various parts of the States continued to appear after this. In 1881, the Annual Report of the National Board of Health for the United States gave a return showing that in the chief cities of the States, with a combined population of 8,533,250, as many as 2,914 deaths, a rate equal to 0.34 of the population, were referred to cerebrospinal fever during 1881. A similar return issued for 1884 showed a somewhat reduced number of fatal cases: in the chief cities of the States, with a combined population of 10,246,695, 1,503 deaths (equal to a rate of 0.15 per 1,000), had been certified during the year. Since that time it has been observed that cerebrospinal fever has been relatively more frequent in the cities than in the rural districts. The disease has seemed to maintain itself in New York for a number of years. The monthly bulletins with regard to infectious diseases are available for the State of New York from 1884 onwards. The annual figures are therefore obtainable from that year till the present day. The subjoined Table shows the prevalence of cerebrospinal fever in New York for the last fourteen years:—

Deaths from Cerebrospinal Fever from 1884 *to* 1897 *in the State of New York (Population in* 1891, 5,970,000).

1884	1885	1886	1887	1888	1889	1890	1891	1892	1893	1894	1895	1896	1897
289*	446	522	540	490	382	482	687	657	853	502	?	?	538

From the above Table it will be seen that cerebrospinal fever was more than usually prevalent in the State of New York during 1893.

In the State of Massachusetts, the public records show that from 1878 to 1896 the average annual number of deaths from cerebrospinal fever in that State (population, 1890: 2,238,243) was 150: ranging from a minimum of 78 in 1878, to a maximum of 171 in 1888. But in 1897 the disease became epidemic, no fewer than 557 deaths being referred to it. This epidemic formed the subject of an official report, made at the request of the State Board of Health, by three eminent investigators, viz.: 1. Dr W. T. Councilman, Professor of Pathological Anatomy, Harvard University, and Pathologist to the Boston City Hospital; 2. Dr. F. B. Mallory, Assistant Professor of

* Nine months only.

Pathological Anatomy, Harvard University, Pathologist to the Children's Hospital, and Assistant Pathologist to the Boston City Hospital; and 3. Dr. I. H. Wright, Instructor in Pathological Anatomy, Harvard University, Director of the Laboratory of the Massachusetts General Hospital. The report issued by these investigators is one of the most complete of its kind, and it will long be a manual of information for all who wish to make themselves acquainted with the etiology and pathology of cerebrospinal meningitis (9). In the report of the Boston City Hospital for 1897, Dr. Francis H. Williams (10) gives a summary of the symptoms, etc., observed in 71 cases of cerebrospinal meningitis admitted to the hospital during the year. This clinical summary will be found very useful to those who are desirous of studying the leading characteristics of the disease.

A somewhat curious epidemic of paralysis (11) in children occurred in the State of Vermont during the summer of 1894, 120 being attacked. Several adults also suffered, and some died. It was denied that this was an outbreak of cerebrospinal fever, but the notes given of typical cases are quite consistent with a diagnosis of this disease.

The current medical journals in the United States are now frequently giving accounts of cases, or groups of cases, of cerebrospinal fever. For example, in the *Pacific Medical Journal* for June, 1898, Dr. H. G. Brainerd gives details of an outbreak of cerebrospinal fever at Los Angeles, California, which began in February, 1898, and comprised some 45 or more cases.

In Chicago, during 1898, some 38 cases of cerebrospinal fever were reported, with 25 deaths: a mortality of 65 per cent; but it is believed that many of the less severe cases did not come under official notice.

From the number of contributions on the subject of this disease published in medical journals in the leading cities all over the States, one is led to believe that this epidemic malady is familiar to the American medical man, and that at present it receives a good deal of his attention.

From *Canada* less is heard of this disease. Small outbreaks are reported from time to time, but, perhaps, owing to the scattering of the population over so wide an area, there are smaller opportunities for its diffusion, and the outbreaks, when they do occur, are on a smaller scale than in the United States. In 1871 there was an outbreak in Montreal. Deaths, though in small numbers, have been recorded in this city each year since 1885. It

is probable that some of the deaths certified as encephalitis were cases of cerebrospinal fever.

Deaths from Cerebrospinal Meningitis and from "Encephalitis" in Montreal (population 1891, 216,644) ; *from* 1885 *to* 1892.

	1885	1886	1887	1888	1889	1890	1891	1892
Deaths from Cerebro-spinal Meningitis ...	31	20	17	23	18	16	10	19
Deaths from "Encephalitis"	291	218	240	285	341	360	324	232

Little information is obtainable from *South America* or from the *West Indies* regarding the occurrence of outbreaks of cerebrospinal fever there. Davidson (12) states that no epidemics have been observed in *British Guiana*, yet outbreaks have been said to occur among coolies during their voyage from India to the West Indies.

Dr. Bolton Corney's paper (5), already mentioned, shows that cerebrospinal fever was introduced into Fiji in 1876. Sporadic cases occurred after this. Certain fatal cases, variously described as nervous ailments, brain fever, idiopathic tetanus, or as sunstroke, he regarded as having been really deaths from cerebrospinal fever. In 1885, some 128 recognised cases occurred, and of these 90 (or 70 per cent.) proved fatal. Besides these 128 cases there were a number of suspicious, mild, or ill-defined cases, not, however, presenting the marked characters of the disease. Nearly all of his cases had a nasal discharge: herpetic eruptions were common. No purpuric spots were observed.

In *India* the disease is well known to the medical officers of some of the jails. An outbreak, for example, was reported from Alipore Jail in 1885-6. Some of the standard medical works state that cerebrospinal fever is unknown in India. Captain Buchanan, I.M.S., has drawn attention (13) recently to the occurrence of small or pseudo-epidemics in the Indian jails. He gives a recent instance of 17 cases among prisoners, with 13 deaths, which occurred during 1897-98 at Bhagalpore.

Scanty information is forthcoming from Asiatic countries with regard to epidemics of cerebrospinal fever, but in Persia an outbreak is known to have occurred in 1874-75.

Europe has never been entirely free from the disease since its first recognition at Geneva in 1805. Immediately after this date the European countries which appeared to have suffered most were France, Italy, and Denmark; later, the malady invaded, so far as can be learned, nearly every

country in Europe, and also those adjacent countries which have frequent communication with it.

In France, widespread outbreaks were reported, particularly in the South, affecting to a large extent the troops in garrison. A considerable epidemic prevailed from 1839 to 1842, and was specially severe upon the garrison towns of Metz, Paris, Bayonne, Narbonne, Bordeaux, and La Rochelle. Another epidemic of the same kind continued from 1846 to 1850. There were also well-marked outbreaks in 1867-68. In 1872 there was an epidemic of so-called "malignant purple fever" in various parts of France, and further outbreaks in 1880, 1881, and 1882 at a number of garrison towns such as Cherbourg and Castres. A recent pamphlet by Dr. Delvaille (14) speaks of a recurrence of the disease in the garrison as well as in the civil population of Bayonne and neighbourhood during 1897. Published statements lead us to the conclusion that there has been distinctly a prevalence of the disease in France during the last two years. The malady is at the present time attracting a good deal of attention in France, and the current medical periodicals contain frequent reference to the subject. Articles have appeared during 1898 in the *Presse Medical* (16), in the *Bulletin Medical* (17), in the *Semaine Medicale* (18), in the *Bulletin et Mémoires Société des hôpitaux de Paris* (19), and in the *Archives de Médicine et Pharmacie Militaire* (20), and in other journals.

Italy has also furnished numerous examples of epidemics of this disease. The earliest recorded was in Southern Italy, from 1839 to 1845. At Bologna an outbreak occurred in 1873, and in the winter of 1873-74, the disease was prevalent in Rome; from this point it spread over the whole of Southern Italy. In 1883 it appeared in Sicily. The deaths from epidemic meningitis for the twelve years ending December 1896, are placed in the following Table, which serves to show that the disease smoulders on for a time, and then bursts out into epidemic proportions. For instance, after almost complete absence in 1891 and 1892, the malady was extremely fatal to a large number of persons during 1893 and 1894.

Deaths from Epidemic Meningitis in the Kingdom of Italy (population 1890, 30,158,408) during each of the years 1885 to 1896 inclusive.

1885	1886	1887	1888	1889	1890	1891	1892	1893	1894	1895	1896
223...	46 ...	326 ...	93 ...	171 ...	64...	12...	28...	2,004...	1,262...	176...	409

In Denmark, cerebrospinal fever appeared in 1845, and

continued to prevail till 1848, spreading in the meantime to Iceland. In 1873-74 it broke out again in Jutland, but since then no very large outbreaks have been reported. From the official Annual Reports of the Central Public Health Office the following Table has been compiled, giving the number of *cases* reported during each of the nine years ending December 31st, 1896 :—

Reported CASES *of Cerebrospinal Fever in Denmark during each of the last nine years*, 1888 *to* 1896 (*population in* 1890, 2,172,380).

1888	1889	1890	1891	1892	1893	1894	1895	1896
38	47	50	85	141	127	49	28	66

A considerable proportion of these cases occurred in Copenhagen, some of them among the garrison.

Sweden.—In 1854 the disease broke out at Göttenburg. It raged for at least ten years throughout one or another part of the country. From 1854 to 1867 the total number of deaths from cerebrospinal fever, officially reported, amounted to 4,577. For the ten years ending 1886, the average number of *cases* reported annually was only 85, but during 1893 no less than 400 were reported. The Table below, compiled from the official Annual Reports of the Central Health Office, shows the number of reported cases for each of the nine years ending December 1896.

Table showing the Reported CASES *of Cerebrospinal Fever in Sweden from* 1888 *to* 1896 (*population in* 1890, 4,784,941).

1888	1889	1890	1891	1892	1893	1894	1895	1896
117	196	605	227	102	111	122	96*	165†

The information obtainable regarding *Norway* is meagre. It is, however, asserted that the disease has only twice been epidemic in that country, but sporadic cases continue to be reported from time to time.

Russia.—Few records are to hand concerning this country. The difficulties of the language render it far from easy to extract from the official reports any facts or figures relating to cerebrospinal fever. Of late, the headings in the official tabular returns of deaths from certain diseases have been also printed in French, but only for the more common zymotic diseases. If the names of all the diseases in the statistical tables were also given in French, the monthly and yearly tabular statements of mortality would become more generally available to epidemiologists, at least in this country.

* Of these 59 died. † Of these 74 died.

In 1863 there was an epidemic in the Government of Kaluga, and in 1865 in the Governments in Poland, in Minsk, as well as in St. Petersburg. Early in 1866 numerous cases were reported from Moscow, and, from 1867 to 1869 in Kalisz. In Southern Russia there was an epidemic affecting the Caucasus, in 1867 and 1868; it spread over the whole of the Crimea. In 1879 it attacked Plozk, Siedlice and Plonsk. For the reasons above stated, I am unable to give any tabular statement as to deaths in recent years.

Roumania suffered in 1869; *Greece* was attacked in 1863; in 1868-69 the whole of the *Ionian Islands* were invaded. In 1887 and 1888 cerebrospinal fever was widely diffused in epidemic form on the shores of the Mediterranean, more especially in *Cyprus*, *Samos*, *Crete*, *Athens*, *Trieste*, *Istria*, and *Sardinia*. An interesting account of this prevalence, from the pen of Sir Richard Thorne, will be found in the Annual Report of the Medical Officer to the Local Government Board for 1888.

Austro-Hungary.—Little was heard of outbreaks of cerebrospinal fever in Austro-Hungary in the first half of the present century. A number of cases were reported from Vienna in 1863. In Galicia, in 1879, there was an epidemic. In 1889 cerebrospinal fever was prevalent in Dalmatia, Istria and Steiermark. In Dalmatia, in 1889, 203 cases were reported, and of these 132 were fatal. In Istria during the same year, 44 cases, with 26 deaths, occurred; and in Steiermark 255 cases, with 106 deaths, were reported. In 1892, in view of the occurrence of a number of scattered outbreaks of the disease, the Government of Austro-Hungary, at the suggestion of the Superior Sanitary Council, made the notification of cerebrospinal fever compulsory throughout the Empire. In 1894 the official returns show that 387 attacks, with 181 deaths, were reported as having been certified throughout the country. In 1898 the disease reappeared in Steiermark at Trefail. An account of this outbreak was published in the official organ of the Central Health Department, *Das Oesterreichische Sanitätswesen*, Nos. 17 and 25, for 1898. It appears that when this outbreak began there were occurring in the district a large number of cases of so-called influenza; and that, along with these, the well-marked cases of cerebrospinal fever occurred side by side. Some doubt was expressed as to the real nature of the epidemic, and a Government Medical Commission, consisting of a Professor of Clinical Medicine, a county medical officer, and a district medical officer, were

instructed to visit Trefail to decide what was the malady in question. The Commission had the further assistance of two local practitioners, and later on had the valuable cooperation of a skilled bacteriologist from Vienna. The epidemic began in February, 1898, in Trefail, which has a population of about 9,000, engaged chiefly in the coal-mining industry. From February to May, 59 cases of cerebrospinal fever were notified; and of these 33, or nearly 60 per cent., died. No figures are published as to the precise number of the so-called influenza cases, but they were said to be numerous. Later on, they came to be regarded as mild or abortive cases of the epidemic cerebrospinal fever. The Medical Commissioners made several official post-mortem examinations; they visited patients who at the time were passing through the acute stage, and others who were in the convalescent stage of the fever, and they came finally to the conclusion that the disease in question was undoubtedly true epidemic cerebrospinal meningitis. Material was also collected for bacteriological examination, and the diagnosis was thereby further confirmed. Scattered cases of the same kind were reported in some of the communes round about Trefail. This outbreak is most interesting from an etiological point of view, more especially in its connection with the prevalence of the so-called influenza, which was not the real epidemic influenza, but a pseudo-influenza due to the infection of cerebrospinal fever.

Germany.—Von Ziemssen states that epidemic cerebrospinal meningitis first appeared in Germany in 1822. There are further references in published medical records from 1834 to 1843, of epidemics of "encephalitis" and of "acute hydrocephalus," which were in all probability epidemics of cerebrospinal fever. From 1863 to 1870 the disease was widely diffused over the eastern and western parts of Germany, attacking isolated localities. These outbreaks were sometimes limited almost entirely to barracks, to workhouses, or to prisons; the populations outside these establishments suffering only in a minor degree. One of the largest and most widely-known epidemics of cerebrospinal fever occurred in 1865-66 in the Province of Dantzic. An account of this epidemic by Prof. Hirsch (1) was read before this Society, as has already been mentioned. The outbreak was further investigated by Dr. Burdon Sanderson, who was sent to Germany for the purpose by the Privy Council. The conclusions he arrived at are contained in the Eighth Annual Report of the Medical Officer

to the Privy Council. This report gives a full description of the clinical characters of the disease, and records of the post-mortem appearances in fatal cases. It is said that, in the Province of Dantzic at least 1,900 persons were attacked, and that about 1,000 of them died. The larger proportion of the fatal cases were children. After this severe epidemic had spent itself, the disease continued to manifest itself up and down the country in scattered groups of cases. It is asserted that the disease became "domesticated" in Germany from this time. In the district of Oppeln, cerebrospinal fever cases were observed in 1876, again in 1879, and still again in 1883 and 1885. During 1886 it began to develop epidemic proportions, and spread to adjoining districts. Throughout 1887 the prevalence continued, 317 cases being reported during that year, with 89 deaths. As is now a common experience in cerebrospinal fever outbreaks, there were noted, in Oppeln, simultaneously with the well-marked cases a considerable number of minor instances in which the diagnosis was to some extent doubtful, but which medical opinion regarded as mild, irregular, or abortive cases of the epidemic malady. Cerebrospinal fever is now included among the infectious diseases of which notification is compulsory in Germany.

In Bavaria cases have been reported from a number of localities, though in no great amount in each year. The subjoined Table gives the reported *cases* in the kingdom of Bavaria from 1887 to 1895:—

Table showing the number of Reported Cases of Cerebrospinal Fever during the last nine years in Bavaria (population 1891, 5,594,982).

1887	1888	1889	1890	1891	1892	1893	1894	1895
54	267	250	228	231	305	230	307	155

During recent years there have apparently been no widespread epidemics in Germany, but rather a number of small groups of cases in different localities. In 1893 an outbreak occurred among the military stationed at Karlsruhe, extending to the garrison at Stuttgart in 1894; and cases continued to occur at the latter place up to 1895, among the troops and also among the civil population. This outbreak was most carefully investigated by Staff-Surgeon Dr. Jaeger, (31) to whom belongs the distinction of having done much to elucidate the etiology of the disease. This investigation will be referred to later on.

During 1894-95, and 1896, the number of small outbreaks appears to have been increasing. In large towns such as Hamburg, for example, there was a perceptible increase of

cases, though no actual epidemic developed. The following Table gives the number of reported cases and deaths in Hamburg from 1884 to 1896:—

Table showing Number of Reported Cases and Deaths from Cerebrospinal Fever from 1884 *to* 1896 *in Hamburg (population in* 1890, 622,530).

	1884	1885	1886	1887	1888	1889	1890	1891	1892	1893	1894	1895	1896
Cases ...	—	49	14	19	9	9	3	9	5	8	8	28	30
Deaths...	—	30	7	14	5	2	2	6	5	7	6	21	26*

The preceding short sketch of the distribution of cerebrospinal fever in foreign countries does not assume to be in any way complete; but the examples given show the wide diffusion of the disease and its frequent occurrence even in recent years in neighbouring countries. But what is of more immediate concern to ourselves is the incidence of this disease in our own country. I now give briefly a short account of what is known on this matter.

Ireland.—The first reported outbreak of cerebrospinal fever in Ireland was in 1845 and 1846. The disease was stated to have broken out at Dublin among the Irish Constabulary, and cases at about the same time also appeared in certain Irish workhouses, including Dublin, Bray, and Belfast. A few cases were again seen in Dublin in 1850. In 1866 a severe epidemic appeared in Ireland. Dr. Mapother, in his account of the outbreak, says that in Dublin 71 deaths were certified from this cause during 1866; and in the adjoining districts of Tullamore, Parsonstown, Mitchelstown, Rathcoole, and Clondalkin some 13 more deaths were recorded. The mortality can only be approximated, as the disease was certified under a variety of names. In all probability the numbers were much greater than stated above. The disease attacked the military stationed in Ireland during 1866. Cases were reported from the different barracks in Dublin, as well as from other parts of Ireland, such as the Curragh Camp. Some cases occurred among the troops forming a flying column engaged in repressing the Fenian rebellion. Staff-Surgeon Marston gives a list of 27 recognised attacks among soldiers and their families; 22 out of the 27 died. This excessive mortality suggests that the less severe cases were not included in the list of attacks. The outbreak of 1866 continued to smoulder on in Ireland during 1867-68, and 1869. As late as 1869 deaths continued to be certified in Dublin from

* In 21 of these, post-mortem examination was made and the diagnosis confirmed.

cerebrospinal fever, cerebrospinal arachnitis, and purpuric fever. At Dundalk, during the first quarter of 1869, 23 cases of cerebrospinal fever were reported, with 11 deaths. In addition to these 11 deaths, the Registrar stated that at least four more deaths could be ascribed to the same cause, though certified under other names. During the second and fourth quarters of 1869, a few other deaths were reported from cerebrospinal fever in Dundalk and in adjoining districts. In Dublin again, in 1885 and 1886, an epidemic of cerebrospinal fever appeared. During 1885, 52 deaths were certified from this cause in Dublin, and in 1886, 28 others. During the first quarter of 1887, three more deaths were certified from cerebrospinal fever, making a total of 83 certified deaths from the disease in Dublin during this prevalence. It is believed that other deaths from the same cause were certified under other headings. During this period typhus fever was certified to have caused 117 deaths, and under the class of "simple continued and ill-defined forms of fever" 97 others were recorded. The subjoined Table shows the fever deaths in Dublin for the four years 1884 to 1887:—

Table showing Deaths from Fever for each of the four years 1884 *to* 1887 *in Dublin (population of Dublin Registration District, estimated to middle of* 1885, 353,082).

—	Cerebro-spinal Fever.	Typhus.	Typhoid.	Simple, Continued, and Ill-defined forms of Fever.
1884 ...	—	83	134	38
1885 ...	52	54	144	39
1886 ...	28	39	129	25
1887 ...	3	24	135	33

There is no authentic information, so far as I can learn, as to any recent epidemics of the disease in Ireland.

Scotland.—It is stated in various text-books of medicine, *e.g.*, in Hilton Fagge's *System of Medicine*, that Scotland has remained free from any outbreaks of cerebrospinal fever. In 1884, however, Dr. Frew, of Kilmarnock, at that time practising at Galston, Ayrshire, read a paper before the Glasgow Pathological and Clinical Society, drawing attention to the occurrence of the disease in Scotland, giving an account of a localised group of cases that had occurred in his own practice. In 1888 Dr. Frew

further reported on the incidence of the malady in Scotland. And again, at the annual meeting of the British Medical Association at Carlisle, in 1896, Dr. Frew read a paper on the same subject, in which he stated that he had collected notes of upwards of one hundred cases of cerebrospinal fever that had occurred in Scotland. These communications, as is often the case under similar circumstances, had the effect of calling forth the experience of other observers, who corroborated Dr. Frew's statements, recalling groups of cases that had occurred within their own practice. Among others who published their experience was Dr. T. J. Maclagan, of London, who formerly practised in Dundee. This paper was published in the *Edinburgh Medical Journal* for 1886, and gave details of some thirty-four cases which came under his notice in 1877-78. It is worthy of mention that the cerebrospinal fever cases sent to the Dundee Hospital were sent in as cases of typhus or typhoid, and were so certified by the practitioners who sent them to hospital. Post-mortem examination of fatal cases showed no evidence of these fevers, but gave conclusive evidence of cerebrospinal meningitis. For example: two brothers, with similar symptoms, were sent into hospital certified to be suffering from typhoid fever. One of them recovered, the other died. Post-mortem examination showed no traces of typhoid fever, but only those of cerebrospinal meningitis. In the *Lancet* for February 12th, 1887, Dr. Sinclair, of Dundee, refers to the occurrence of the disease in that town, and gives particulars of a necropsy on a lad admitted to hospital as typhus, but who died on the fourth day of his illness. The post-mortem appearances were those of cerebrospinal meningitis.

Dr. Frew has been kind enough to inform me privately of a number of instances where medical men have written to him as to cases which were undoubtedly cerebrospinal fever. Some were mentioned—for example, where children became permanently deaf after a febrile attack—which could not at the time be attributed to any specific infection, but which were now attributed to cerebrospinal fever by the writer. It seems evident then, that though, till recently, no recorded outbreaks were forthcoming from Scotland, yet that country cannot claim immunity from the disease. As Dr. Frew puts it, "Where the disease is not expected it is not looked for," and consequently passes unrecognised and undetected. The same remark would seem to be applicable to other countries besides Scotland.

The appended Table gives, for thirteen years ending 1895, the cases certified to have died in Scotland from cerebrospinal fever; it also gives the deaths from certain diseases which are sometimes confused with cerebrospinal fever.

Table showing Deaths certified from Cerebrospinal Fever and certain other Diseases in Scotland, for the thirteen years 1883 *to* 1895 (*population* 1891, 4,124,691).

—	1883	1884	1885	1886	1887	1888	1889	1890	1891	1892	1893	1894	1895
Cerebrospinal Fever ..	7	9	8	10	4	6	2	3	6	5	5	2	6
Idiopathic Tetanus ..	48	52	48	44	48	46	18	38	35	38	71	50	57
Purpura	52	46	44	38	57	66	52	45	45	46	55	47	51
Inflammation of Brain ..	1503	1688	1727	1753	1628	1588	1031	1614	1684	1546	1534	1438	1659
Tubercular Meningitis ..	1071	1725	1219	1249	1224	1186	1163	1140	1242	1112	1185	1150	1254
Influenza	67	50	55	55	51	46	59	830	2387	1786	1248	812	1752

England and Wales.—The early records of the disease in England are very scanty. An outbreak in 1807, reported from Devonshire, and another from Sunderland in 1830, are believed now to have been cerebrospinal fever. In 1850 cases were reported from Liverpool and from Rochester. The medical journals contain references, after this, to scattered cases in London and the Provinces, especially during 1856, 1858, 1859, 1865 and 1866. The occurrence of the severe outbreak in Eastern Prussia in 1865 called public attention to the disease in this country, and some cases were reported during 1866 in London, and again at Rochester. In the same year a well-marked epidemic occurred near Lincoln, at Bardney, (26) a village of some 1,500 inhabitants. In 1876 there was a somewhat widespread prevalence of the malady in the Midlands. Foster, (25) of Birmingham, saw eight fatal cases in his practice. Other groups of cases were reported from Bath, Plymouth, and Oxford: in the last-named place, two of those attacked were young militiamen. In the winter of 1885-86 a typical outbreak was reported at Devonport among the garrison, and also among the men on board some of the warships in the harbour (21). In the same year a fatal case occurred in the barracks at Chester. In the medical journals, early in 1887, a number of observers related, under various designations, cases with cerebrospinal symptoms which had come under their notice: the characters of the cases and the post-mortem

appearances suggest strongly that the patients had suffered from cerebrospinal fever.

In a paper published in the *Lancet* in March, 1895, Dr. Ormerod (22) gives an account of ten fatal cases, as well as four non-fatal instances, which had occurred in St. Bartholomew's Hospital during the time he was Registrar there, from 1887 to 1893. He excluded from his list a number of other cases in which any recognised cause of the disease could be found, such as tubercle, injuries to the head, otitis, disease of the cranial bones, ulcerative endocarditis, pneumonia, erysipelas, etc.

In 1890, the occurrence of cases of cerebrospinal meningitis was mentioned in the reports of Medical Officers of Health in the counties of Cambridge and Monmouth. During the same year cases were reported from Leeds (8) and from London. In the autumn a well-marked prevalence of this disease came under my own notice in the eastern counties (22). In 1891, small groups of cases were reported from Shropshire, Hertfordshire, and Lincolnshire. In the latter county, in the Market Rasen district, which on one side adjoins the Lincoln Rural district, there occurred a curious combination of what appeared to be—or what was locally considered to be—epidemic influenza along with epidemic cerebrospinal meningitis. The influenza cases began to appear among adults in December, 1890, but the severe cerebrospinal fever cases did not begin to occur among children until June. The children attacked had marked retraction of the head, and some seventeen of them recovered with paralysis of the limbs, with a squint, or with deafness. Three cases died, and on one of the latter a purpuric eruption had been noticed. This outbreak resembles in many respects the epidemic already mentioned which occurred at Trefail, in Steiermark, during 1898, where the influenza-like illness, after careful inquiry, was adjudged to have been due to cerebrospinal fever infection and not to epidemic influenza. In the same year (1891), an epidemic affecting Raunds, Heyford, and other localities in Northants was investigated by me, in which suspicion arose that the anomalous symptoms observed were due to cerebrospinal meningitis (23), though it has to be admitted the majority of the cases had ill-defined symptoms. It is however, possible, that this outbreak was not true cerebrospinal fever, but was similar in character to outbreaks which occurred in the Transvaal in 1894 and in 1898, mention of which is made further on (38). So far as I can learn, no special prevalences of cerebrospinal fever were

noted in 1892; but I heard from a number of country practitioners that they had noticed in their practices during the year cases which puzzled them. For example, one practitioner in Notts mentioned that he had seen about twelve children attacked by an illness which was marked by headache, vomiting, stupor, pain and stiffness in the neck, passing in some instances to retraction of the head, inability to stand or walk; in a few, some purpuric spots had been noted. One girl died after twenty-four hours' illness from uncontrollable vomiting of blood. A few developed an ear discharge, and some had violent epistaxis. Another practitioner in the same county spoke of having had, in 1891, a number of cases with spasm of the muscles of the neck, retraction of the head and conjunctivitis. Five other practitioners in the county of Notts told me of similar cases, or groups of cases, fatal and non-fatal.

In 1893 I heard of groups of cases of cerebrospinal fever which had been noted in Wiltshire and in Essex. In 1894 my attention was called to another suspicious outbreak among children in a rural district in Suffolk, adjoining the locality where a pronounced group of cerebrospinal fever cases had occurred, and which I investigated in 1890. This outbreak in the Suffolk rural district had many characters which aroused suspicion that cerebrospinal fever was again in question (24). In 1895, a small outbreak was reported from a Lancashire district by the Medical Officer of Health in his annual report. In 1896, no information reached me of any further outbreaks during that year. In 1897, I had again to investigate a reported epidemic in Lincolnshire, at Messingham, in the Brigg rural district. This district lies not far from Market Rasen, where an epidemic occurred in 1891. At other points in the Brigg rural and other adjoining districts in North Lincolnshire, I heard of cases strongly resembling cerebrospinal fever which had occurred during 1897, as well as in some of the previous years. I saw, for example, one case just past the acute stage, and which had been seen by three medical men who had agreed that the case was "influenza with cerebrospinal meningitis." This patient had had the usual train of symptoms, including retraction of the head and a purpuric eruption. He had been in a state of coma, passing his urine unconsciously in bed, and had gradually emerged from this dangerous condition in a state of great debility and emaciation. Dr. Eminson, of Scotter, a well-known practitioner in North Lincolnshire, gave me notes of a number of scattered cases that had been noticed in his

practice prior to the Messingham outbreak. Other practitioners spoke of fatal cases that had had delirium, retraction of the head, and coma. Some so-called influenza cases had developed a purpuric eruption. Altogether, some nine or ten medical men in North Lincolnshire testified to having seen cases of a suspicious nature resembling cerebrospinal fever. Lastly, in 1898, information reached me of a curious outbreak in a rural village in Buckinghamshire, where, in a population of 300 persons, thirty were attacked and three died, from an illness that was of an anomalous kind; and although the cases were examined by four medical men, the exact nature of the epidemic was not settled. The written account sent to me excited suspicion in my mind that this might have been another of the anomalous forms of cerebrospinal fever that have been elsewhere observed. Unfortunately, owing to other engagements, I was unable to visit any of these last-named cases, and no post-mortem had been held on any of the fatal cases.

Before passing to discuss the etiology of this disease, I give, in the subjoined table, the deaths certified in England and Wales from cerebrospinal fever from 1877 to 1896 :—

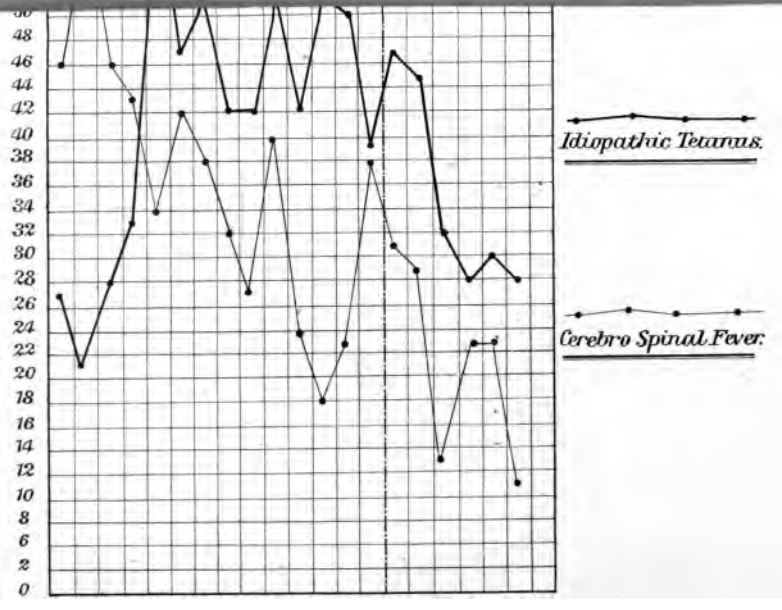
Population of England and Wales 1891, 29,002,525.

1877	1878	1879	1880	1881	1882	1883	1884	1885	1886
46	58	46	43	34	42	38	32	27	40

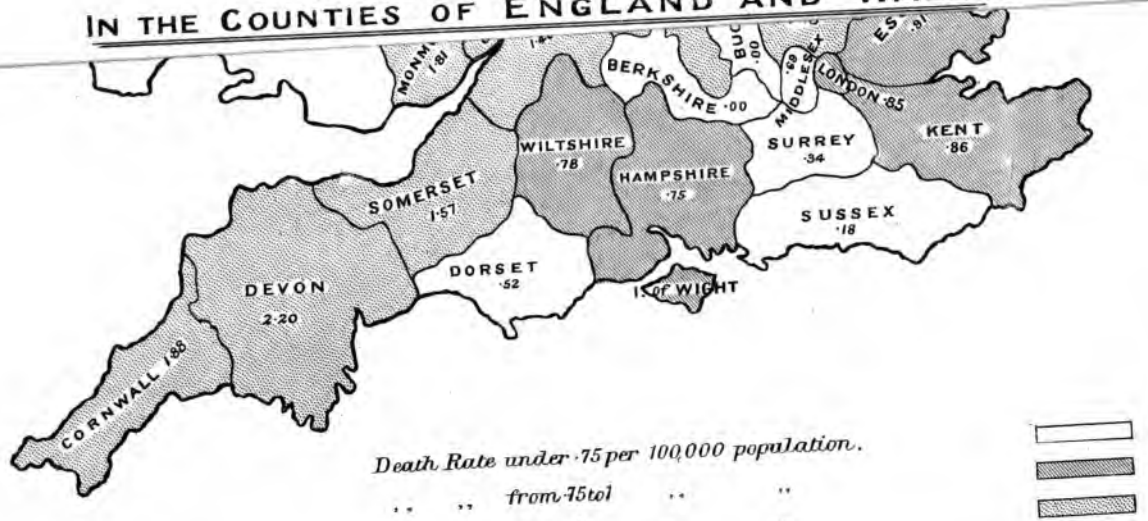
1887	1888	1889	1890	1891	1892	1893	1894	1895	1896
24	18	23	38	31	29	13	23	23	11

There is reason to believe that the above table does not fully represent the total number of deaths which have resulted during the last twenty years from cerebrospinal fever; and that deaths from this cause have been classed under other headings, *e.g.*, inflammation of the brain and its membranes, or tubercular meningitis, idiopathic tetanus, influenza, sunstroke, &c. With regard to sunstroke I may mention that within my own experience I have known of a youth suddenly attacked in the fields with what afterwards turned out to be cerebrospinal fever. The illness at first was attributed to a sunstroke, though it was a cloudy day and actually raining when the patient fell down. The appended chart shows the curves of mortality, during the last twenty years in England and Wales, of inflammation of the brain, tubercular meningitis, idiopathic tetanus, and cerebrospinal fever. The appended map shows roughly the incidence of cerebrospinal fever upon the counties of England and Wales.

DEATHS IN ENGLAND AND WALES
FROM CEREBRO SPINAL FEVER, TUBERCULAR MENINGITIS, INFLAMMATION OF BRAIN & IDIOPATHIC TETANUS, 1877-1896.



DEATH RATES FROM CEREBRO SPINAL FEVER FOR THE PERIOD 1886-1896.
PER 100,000 POPULATION.
IN THE COUNTIES OF ENGLAND AND WALES.



The notes of outbreaks of the disease that have come to my notice during the last few years lead me to think that cerebrospinal fever is not such a rare disease, after all, in England as has been supposed; and that it passes under other designations, especially as influenza, not only in this country but in the United States, Germany, Austria, and elsewhere.

The report on the outbreak at Trefail, in Steiermark, already alluded to, states that during the last few years groups of cases with catarrhal symptoms, with or without nervous complications, have become of somewhat common occurrence; and that in a number of these local epidemics bacteriological investigation has proved the presence of the specific organism of cerebrospinal fever, to which reference will be made when dealing with the etiology of the disease.

There seems recently to have been a tendency to attribute any unusual or undiagnosed form of illness to influenza infection. Of late years cases of suppurative meningitis, following influenza, have been published in the medical journals. Other similar cases are stated to have developed tetaniform convulsions, and a few have been followed by paralysis. It appears either (*a*) that cerebrospinal meningitis is becoming a more common complication of epidemic influenza than formerly; or (*b*) that the two infections are not unfrequently found in an epidemic form running concurrently; or (*c*) that cases of epidemic cerebrospinal meningitis are frequently mistaken for cases of epidemic influenza. Some authorities, indeed, have advanced the view that these two diseases are one and the same. But it is now generally admitted that epidemic influenza is associated with a special organism, the bacillus of Pfeiffer; and, as will shortly be seen, cerebrospinal fever is now known to be associated with a special diplococcus. So that to the Bacteriologist in future we shall have to look to settle the diagnosis in doubtful cases.

Instances are frequently reported of recoveries from tubercular meningitis. It is admitted that in some cases the clinical characters are difficult to distinguish. Hitherto tubercular meningitis has been regarded as practically an incurable disease, and the occurrence of recovery has been looked upon as a proof that the diagnosis has been wrong. Some cases tested by means of lumbar puncture have shown the presence of both the tubercle bacillus and the diplococcus of cerebrospinal fever, a mixed infection.

Etiology.

For a long time the etiology of this disease has remained obscure. It is only within the last few years that light has been thrown upon it. Almost from its first recognition as a specific disease it had been noted that epidemics of cerobrospinal fever showed a strong tendency to attack soldiers. Many of the early outbreaks were entirely confined to the garrisons. Hirsch (27), for example, mentions that, of 62 epidemics recorded in France, in 43 the outbreak was absolutely confined to the troops, in 6 other epidemics the military suffered chiefly but the civil population were occasionally attacked, while in 5 others the disease affected soldiers and civilians in equal amount. The same experience has been noted in Germany, and, on a smaller scale, in America. In our own country the military have not escaped. The Irish Constabulary, practically a military body and living in barracks, suffered in 1845 and 1846. Again, in 1866 and 1867, the soldiers in the Dublin barracks and in the Curragh Camp, as well as the troops forming the flying column dealing with the Fenian rising, were attacked by cerebrospinal fever. In 1885-86 the garrison at Devonport was attacked.

Next to the soldiers, those who were most apt to be attacked were the inmates of public institutions, such as prisons and workhouses. In India it is regarded as a disease of jails, and some outbreaks recorded in Europe and America have been confined to convicts. In Ireland, workhouses have suffered in several of the epidemics. In 1863 a large orphanage was the scene of a smart outbreak in Vienna. The above remarks suggest that the disease is apt to spread among those who are housed together in more or less crowded apartments with deficient ventilation. *Fatigue* is by some regarded as a predisposing cause.

Season.—This disease shows a preference for developing epidemics during the winter and spring, at a time when conditions of overcrowding and lack of proper ventilation would be most likely to occur. Some have attributed this seasonal occurrence to coldness or dampness of weather causing the closing of windows and doors, and the consequent diminution of the purity of the air of the apartments.

Age Distribution.—Cerebrospinal fever shows a marked disposition to attack the young. In the epidemic in the Province of Dantzic in 1865, Hirsch states that in the two circles of Berent and Karthaus 779 persons died, and of these, 737, or 93.3 per cent., were under 15 years of age.

It has, however, to be remembered that very young subjects die more readily from this disease than older persons, and that for this reason the mortality is not always to be taken as a test of the age incidence. On the other hand, attacks in very young children are more easily overlooked. It must be admitted that youth and infancy predispose to cerebrospinal fever.

Infectivity.—For a time it was sought to prove that epidemics of this disease arose from some common article of diet, and not from infection. Diseased or mouldy grain was suggested by the late Sir B. Richardson; and that this acted as in ergotism. Others suggested marsh miasm, and some spoke vaguely of telluric influences (21). No satisfactory proofs, however, were ever adduced in support of these theories. For a long time even the identity of the disease was denied. It was urged that it was only a variety of typhus, and that its etiological conditions were those of typhus. One of the most strenuous supporters of this view was the late Dr. Murchison. Others, again, while admitting that it was a separate disease, denied that it was communicable from person to person. I believe that Dr. Burdon Sanderson held this view as a result of his visit to East Prussia in 1865. On the other hand, authorities like Professor Hirsch and Sir John Simon, while admitting that the malady did not spread like small-pox and scarlatina, yet hesitated to support the theory of incommunicability.

For the last twenty years it has been felt that, if the disease was spread by infection there must be a special organism associated with it; and search accordingly began to be made for the specific microbe.

In 1881 Gaucher (28) stated that he found micrococci in the urine and blood of a patient suffering from epidemic meningitis, as well as in the exudation taken after death from the pia mater of the brain and spinal cord. In 1883 Ughetti (29), stated that he had found micrococci in the exudation and in the blood. In 1884 Marchiafava and Celli (30) found micrococci in a case of epidemic meningitis after death. The organisms were in the intracellular fluid and in the colourless corpuscles within the affected part. In 1886 Leichtenstern found, during an epidemic at Cologne, in the exudation in the meninges a few cocci, sometimes single sometimes in groups, similar in arrangement to gonococci, enclosed in white corpuscles. In 1887 Weichselbaum (33) described a peculiar form of micrococcus which he had found in six cases of acute cerebro-

spinal meningitis. Though Weichselbaum identified and described this diplococcus intracellularis meningitidis in 1887, no further confirmation of his discovery took place till Jaeger's investigations in 1895. Meanwhile, Fraenkel, Netter, and others identified a microbe in cases of croupous pneumonia. This organism received the name of the Pneumococcus Lanceolatus of Fraenkel. For some years this pneumococcus of Fraenkel was erroneously recognised as the organism always associated with cerebrospinal fever. It is admitted that in certain cases of epidemic meningitis the pneumococcus is found in the brain exudation, but Jaeger, in 1895, made investigations during an epidemic at Stuttgart, which proved that the diplococcus of Weichselbaum is the true cause of cerebrospinal fever, and that the pneumococcus may be also present as a secondary or mixed infection, the same being true of streptococci, or even of tubercle bacilli. One cannot deny that epidemics have occurred in which both pneumonia and cerebrospinal meningitis have been present, and that such epidemics have been proved to have been due to infection by the pneumococcus of Fraenkel; but it is maintained that these epidemics are not those of true cerebrospinal fever. An interesting example of this kind of outbreak occurred in the Transvaal in 1894, and again in 1898 (38). The pneumococcus is known to be a ubiquitous organism, and its frequent presence in meningitis epidemics as a secondary infection is not at all remarkable. In true epidemic cerebrospinal meningitis, the organism that is invariably present is the *diplococcus* (sometimes called the *tetracoccus*) *intracellularis* of Weichselbaum. This organism has been found in the exudation on the surface of the brain and spinal cord after death. It has been cultivated outside the body on various media, such as Agar-Agar. In cultures the organism does not give a profuse growth on any medium, but the American observers have found it to grow best on the blood serum mixture of Loeffler as prepared by a method devised by Mallory.

Jaeger's conclusions have been confirmed by Heubner, Holdheim, Petersen, and others on the Continent; and by Councilman, Mallory and Wright, as well as by others in America. The organism has been found, as has been said, on the surface of the brain and spinal cord, and in some cases in the fluid in the ventricles. It has been obtained during life by lumbar puncture from the spinal canal.

Heubner (34) was the first to find it in this fluid during

life. The organism is found in largest amount during the first week of the attack. When cases survive the illness for some time, the organism disappears, and cannot be discovered in the cerebrospinal fluid nor in the exudation on the surface of brain and cord. The organism has been injected into animals such as rabbits, mice, and guinea-pigs without result, but injection of infectious matter into the spinal canal of the goat produces rapid death, and the organisms can then be obtained from the exudation on the surface of brain and cord.

The organism has been found in the nasal discharges of patients suffering from cerebrospinal fever during life as well as after death. Among a number of examples of this published in medical journals, one may mention that in the epidemic of 1898, at Trefail in Steiermark, already referred to, Dr. Ghon, of the Vienna University Pathological Anatomical Institute, found Weichselbaum's diplococcus intracellularis not only in the exudation on the surface of the brain, but also in the nasal secretion of living patients.

Schiff (37) examined the nasal secretion in twenty-seven cases in which the nose was either healthy or the seat of only a slight catarrh. In seven out of the twenty-seven he found the diplococcus intracellularis present, but in only three cases did it give rise to a pure culture. Heubner also found it in the nasal cavities of a person suffering from tubercle, so that here was a possibility again of a mixed infection. This fact that the organism is found in the nasal cavities suggests the way in which infection may enter the system. For a long time it puzzled observers to account for the passage of infection from one case to another. The seat of the disease was within a bony cavity, from which there was no direct communication with the outer air. But since the organism has been found in the nasal cavity, it has been concluded that this is its first point of entry to the system, and that it penetrates to the meninges, possibly finding its way to the brain through the ethmoidal cells. The organism has sometimes been found in the secretion from the tonsils, and more rarely in the expectoration from the lungs.

Jaeger thought that the infection occurs always through the nose, and that the presence of the diplococcus can establish the diagnosis.

The organism is said, when cultivated outside the body, to be of feeble vitality. When the cultures are kept in a room at ordinary temperatures they die in a few days,

but when kept in an incubator and protected from drying up, they may live eighty days or more, retaining their virulence. But it has to be remembered that we cannot produce in the laboratory the precise conditions that the diplococcus finds in Nature.

The organism is able to enter the lung and cause pneumonia. When it finds a foothold in the throat it produces tonsillitis, which has been found a marked feature in some cerebrospinal fever epidemics.

The nasal discharges upon handkerchiefs or linen may convey infection, or the nasal discharges aspirated into the throat, as is the custom of some, and then spat out upon the ground, becoming dried, and perhaps thereafter wafted by the air, may finally reach the nasal mucous membrane or throat of a susceptible subject. This expectoration of infected sputum may furnish the means for spreading this disease in much the same way as tubercle is sometimes spread. There is reason to believe that many instances of sporadic meningitis are due to the *diplococcus intracellularis*. From such sporadic cases, under conditions which have not yet been fully established, it is probable that epidemics are gradually evolved.

An interesting question has recently arisen respecting the relationship of the simple meningitis of infants to epidemic cerebrospinal meningitis.

For some time there has been a growing impression among specialists that the so-called "infantile paralysis" is due, after all, to an infection. Dr. Buzzard supported this view in an address, delivered at the last annual meeting of the British Medical Association in the Section of Neurology, "On the Influence of Micro-Organisms and their Toxines in the Production of Diseases of the Central and Peripheral Nervous System." Dr. Still, in the *Journal of Pathology and Bacteriology* (No. 2 for 1898) contributes a paper on "Simple Posterior Basic Meningitis of Infants:" and again, in the *British Medical Journal* for October 15th, 1898, he writes on the same subject, and expresses the opinion, based on post-mortem examination and bacteriological investigation, that this infantile ailment, the simple posterior basic meningitis of infants, is a specific disease due to an infection associated with a particular micro-organism. He believes this organism, which he has isolated and described, to be only a variation or modification of the *diplococcus intracellularis* of Weichselbaum. This interesting and valuable contribution to the etiology of meningitis will doubtless be followed up by other observers as well

as by Dr. Still, and when confirmed and developed it will throw much light upon the question of sporadic cases of meningitis and their relation to the epidemic variety which we have been considering.

Cerebrospinal fever, as a rule, does not appear to cause multiple cases in invaded households, and this fact has been adduced in the past to support the view that the disease was not due to infection. We now know that the disease is due to a specific infection, and we are led to believe that it spreads mainly, as has already been said, by means of the nasal discharges,—for example, by the use of an infected handkerchief, or from dried sputum which has been ultimately wafted into the nasal cavity of a susceptible person.

Ordinarily, no doubt, the mucous membrane of the nasal passages in healthy persons has sufficient vital power to resist the deposited specific organism, and prevent its multiplication, and penetration to the brain. But in susceptible persons this power of resistance is feeble, and the individual therefore succumbs to the infection and develops the disease.

In some epidemics the occurrence of profuse nasal discharge in persons attacked by cerebrospinal fever has been noted. It would be interesting to learn if the presence of profuse nasal discharge in the infected patient increases the risks of infection to the persons coming in contact with him.

Undoubtedly, the infection in some outbreaks appear to have acquired a greater intensity of virulence than in others. But what are the conditions which have aided or produced this increase of virulence has not as yet been determined. Further observation in this direction is desirable.

Cerebrospinal Meningitis in Animals.

Several observers have reported the occurrence of an epidemic among animals coincidently with the prevalence of cerebrospinal fever in man.

In New York, at the end of 1872, there was a very remarkable and fatal epidemic affecting horses. In the six weeks ending December 2nd, no fewer than 1,412 horses are stated to have died from an epidemic disease described as "Influenza." The veterinary surgeon who reported to the State Board of Health on the outbreak, stated that

"the nervous system was affected in a few cases in the form of cerebral or cerebrospinal meningitis. Many of the hard-worked animals were attacked by purpura hemorrhagica, and a large number of the cases terminated fatally." The artist who sketched some of the post-mortem appearances, from preparations recently taken from horses that had died from the epidemic malady, took suddenly ill with pneumonic symptoms and died. It may be remembered that during the same year New York had suffered from a severe outbreak of cerebrospinal fever which caused 782 deaths.

In the Irish outbreak of 1866-67, for a period of eighteen months, it was noticed that "purples" had been rife among pigs. Many diseased carcasses were seized in the markets. Mr. Hugh Ferguson, Veterinary Medical Officer to the Privy Council in Ireland, gave some particulars of the disease. The illness was short, death supervened rapidly, purple blotches were observed on the skin. He states that "purples" were common among pigs in Dublin and Belfast in 1846, during the cerebrospinal fever epidemic of that year. Oxen, too, were found to have died suddenly, bloody effusion being found, after death, upon the brain. In the account of the epidemic of paralysis among children in 1894, in the State of Vermont, Professor Macphail states that twelve horses died, about the time, of what was called cerebrospinal meningitis, but he was unable to obtain any reliable account of the cases.

A Russian authority, Uspenski (36), published in 1896 a case of epidemic cerebrospinal meningitis in a dog. Another foreign veterinary journal (35) gave recently an article on "Infectious Cerebrospinal Illness in Horses." Young dogs and cats are known to suffer from distemper—a term which probably includes a number of infections. In certain instances dogs and cats die from convulsions in the distemper, while others recover with paralysed limbs. It is quite possible that these are instances of cerebrospinal fever in animals, and that if search were made the diplococcus intracellularis would be found in the brain or spinal cavity.

Certain animals do not appear to be susceptible to the infection of the diplococcus intracellularis. Rabbits, mice, and guinea-pigs do not appear to suffer when cultures of the diplococcus are injected into them.

An interesting case is reported, where a young man developed cerebrospinal fever, which was attributed to his having slept for some hours on the top of a cartload of

stable manure, which he was taking from London to a market-garden district in Hertfordshire. This case was the first of a group, and its origin could not be explained by those who investigated it, except in the way above mentioned. This mode of origin suggests that the manure contained the infection, and that it had been derived from an infected animal.

The information at our disposal on the question as to whether or not animals suffer from cerebrospinal fever caused by Weichselbaum's diplococcus, and if so, whether they can transmit the disease to man, or *vice versâ*, is insufficient to enable us to arrive at a definite conclusion. But the experiment upon the goat shows that this animal at least is susceptible, and that after injection of the organism into it the diplococcus can be recovered from the exudation upon the surface of the brain after death.

The prominence which has recently been given to this disease in medical literature shows that an increasing interest is being taken in cerebrospinal fever, and that observers on the Continent of Europe and in America, as well as at home, are giving special attention to the malady. Considering the great advance which has taken place during the last three years in the knowledge of this disease, we are led to hope that before long all that has been dark and mysterious respecting the origin of its epidemics will be swept away, and that the best means for the prevention of the malady will then be formulated, so that its occurrence in epidemic form may in a large measure, if not altogether, disappear, and become only a memory of the past.

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NOTES ON CEREBROSPINAL FEVER.

CONTRIBUTED BY G. F. STILL, M.D., AND J. W. WASHBOURN, M.D., AT THE CONCLUSION OF DR. BRUCE LOW'S PAPER.

DR. G. F. STILL observed that in the extremely interesting paper read by Dr. Bruce Low on Epidemic Cerebrospinal Meningitis, allusion was made to the disease of infants which was known by the name of "Simple Posterior Basic Meningitis," or "Chronic Basilar Meningitis." The data were not yet sufficient to allow of any dogmatic statement being made as to the relation of this form of meningitis to the epidemic disease, but there were, Dr. Still thought, sufficient grounds for at least raising the question of possible identity.

From the clinical standpoint there were several features in which these diseases resembled one another; the symptoms and complications were to a considerable extent the same, and an important point of similarity was found in their seasonal relations: the epidemic disease, as Hirsch had pointed out, was particularly a disease of the winter and the spring, and the seasonal curve of posterior basic meningitis showed exactly the same variation.

In their morbid anatomy there was also much resemblance; both forms of meningitis affected the base rather than the vertex of the brain, and both were sharply differentiated from meningitis of pneumococcal or streptococcal origin, and from secondary meningitis of all kinds, by the absence of any obvious source of infection in the body; the infantile and the epidemic disease were, in fact, alike in being "primary," not secondary forms of meningitis.

The last link in the chain of evidence, which must go far to establish the identity of these two diseases, was their bacteriology. In the case of the epidemic disease, it was now practically certain that the specific micro-organism is the diplococcus intracellularis; while in the simple posterior basic meningitis of infants there was good reason to believe that there was also a specific micro-organism, a diplococcus which, if it were not identical in every way with the diplococcus intracellularis, at any rate must be merely a modification of it.

He thought that it would be seen, from this rough comparison, that there were at least some grounds for raising the question whether the posterior basic meningitis of infants might not be simply a sporadic manifestation of the disease known as epidemic cerebrospinal meningitis; and the question was one of some importance, for the infantile disease occured every year in London in considerable numbers: and it was essential to the study of the epidemic spread of any disease that its sporadic manifestations should be fully recognised.

DR. WASHBOURN thought that the term Epidemic Cerebrospinal Meningitis was misleading. On the one hand, there were other forms of cerebrospinal meningitis which were epidemic, and on the other hand the disease often occurred in the sporadic and not in the epidemic form.

Cerebrospinal meningitis might be of various origin, such as disease of the middle ear, injury to the cranial bones, etc. But whatever the origin, there were three micro-organisms prominent in the causation. These were: the tubercle bacillus, the pneumococcus, and the diplococcus intracellularis. Tuberculous meningitis exhibited clinical symptoms which were sometimes characteristic, but the other forms of meningitis might be indistinguishable from one another. He did not consider herpes of any diagnostic value in distinguishing a pneumoocccal meningitis from a true epidemic cerebrospinal meningitis. In all pneumococcal infections herpes was common. By lumbar puncture and a bacteriological examination of the fluid, it was possible to make a diagnosis. The operation could be performed with an ordinary exploring syringe, and an anæsthetic was not usually necessary. The theca of the cord was punctured below the termination of the cord, so that no danger arose. He did not often perform the operation himself, as he did not consider it of much direct or indirect therapeutic value.

A pneumococcal meningitis might occur in epidemic form. Drs. Brodie, Rogers, and Hamilton had recorded a very interesting outbreak among Kaffirs, in which the virus obviously gained access to the body by the nasal cavity. Dr. Washbourn was satisfied that the micro-organism found by these authors was a true pneumococcus, as both Dr. Rogers and Dr. Hamilton had worked in his laboratory and were familiar with this micro-organism.

The form of meningitis which goes by the name of

epidemic cerebrospinal meningitis is caused by the diplococcus intracellularis. It probably gains access to the body in most cases by means of the nose. The diplococcus intracellularis is a distinct species to the pneumococcus. The latter exhibits a number of varieties; but the speaker, who had had considerable experience in this matter, had never obtained a variety which he could not distinguish from the diplococcus intracellularis. Besides cultural and morphological differences, there was a marked difference in pathogenic properties. The pneumococcus was highly virulent to rabbits, and but slightly virulent to guinea-pigs, while the diplococcus intracellularis was slightly virulent to guinea-pigs, and not at all virulent to rabbits. He believed that sporadic cases of epidemic cerebrospinal meningitis were common in this country. He quoted a case which was under his care last year; but as recovery ensued, a bacteriological examination was not carried out. Dr. Still, however, had recorded a number of cases of posterior basal meningitis which were caused by the diplococcus intracellularis. Dr. Washbourn had examined his cultivations and had made inoculations, and was satisfied that they were the same micro-organisms as produced true epidemic cerebrospinal meningitis.

TEN YEARS' EXPERIENCE OF ENTERIC FEVER IN A MIDLAND TOWN, AND ITS LESSONS.

By PHILIP BOOBBYER, M.B.

(*Read: February 17th*, 1899.)

I HAVE been largely influenced in my choice of this subject by a desire to publish some of the facts stored up year by year in our office registers of infectious diseases; facts which otherwise might never reach the light. There is nothing very new to bring forward, nothing very startling; but still I hope there may be something of interest to other members of the Public Health Service who happen to hear or read what I have to say. Parts of this have already been published, but I have endeavoured here also to bring out other facts which on previous occasions, for lack of time or other reasons, I have been unable to adduce. My principal aim, in dealing with this subject on former occasions, has been to show the definite causal relation subsisting between the accumulation and deposit of organic filth in dense urban districts, and the endemic occurrence of enteric fever therein. This has been strongly illustrated in Nottingham, by the high rate of incidence of the disease upon houses furnished with conservancy closets (especially midden-privies) as compared with those having w.c's. I shall have more to say on this subject anon, but may mention here a few facts respecting the data upon which my conclusions are based, by way of showing to what extent they may be relied upon. The cases and reputed cases are notified in the usual way. The Infectious Diseases Inspectors then visit the houses in which the cases are reported to have occurred, and record answers to a printed schedule of questions respecting the patient or patients and other members of the household, and the house and its surroundings. These answers or reports are brought to me, and if from the circumstances of any case removal to hospital appears desirable, the patient is now usually removed. We have at the present time, and have had for the past two years, pretty ample accommodation for typhoid cases

in the "fever blocks" of our General and Childrens' Hospitals, and in the "typhoid block" of our City Isolation Hospital—some 60 to 70 beds in all being available. The blood test (Widal's test) for diagnostic purposes is applied in hospital when necessary, but it is seldom used before the removal of the patient from the home. Our Infectious Diseases Registers are made up from day to day as the Inspectors' returns come in, and it is from the Enteric Fever Register so compiled that my facts and figures are gathered. It is hardly necessary for me to say that this system of registration did not originate with me. It was in force in Dr. Seaton's time at Nottingham; it was modified and much extended by Dr. Whitelegge; and subsequent alterations in my time have been for the most part only in connection with methods and extent of inspection, number of facts recorded, and other matters of detail. And, further, most of you will be aware that similar records are now made, and have been made for many years past, in many of our large towns.

It may seem to some that, for all practical purposes of Public Health administration and reform at least, we have already as much knowledge as we want concerning the life-history and etiology of enteric fever. This, however, I think I am right in saying, is not the view of this Society, nor of most of my colleagues in the Public Health Service. Notwithstanding the splendid scientific energy expended in this field of inquiry, especially during the past fifty years, we are as yet by no means informed of all the factors constant and otherwise that go to complete the series of causative elements of endemic typhoid fever.

Far be it from me to speak disparagingly of the work done and the advances made during the period I have mentioned. There is probably no chapter in the history of epidemiology more full of interest and importance to the human race than this. From the time when Pettenkofer and his school, at Munich and elsewhere on the Continent, began to direct attention to the soil as probably the principal habitat of the typhoid poison, down to about 1884, when Eberth, Klebs, Gaffky and Coats, between them, had finally prepared pure cultivations of the typhoid bacillus, and thence onward to the present day, when we have the startling confirmation of some of Pettenkofer's opinions afforded by the experiments of Drs. Sidney Martin and John Robertson, can be traced—as usual with most items of human knowledge—the evolution of order from chaos, the slow but steady accumulation and arrangement

of facts which go to broaden, strengthen, and define the mental grasp of various correlated phenomena. I hope I shall not be misunderstood. I do not for a moment suggest that the innumerable instances in which epidemics of enteric fever have been traced solely to the agency of polluted water, milk, etc., in this country, have been examples of incorrect reasoning. I simply mean that the explanations mostly given of the means of convection of the specific poison have not been exhaustive. In my recent studies of the literature of the subject, I have been greatly impressed with such pronouncements as those of Sir Richard Thorne at Birmingham, in October, 1897; of Dr Bulstrode in his report, dated March, 1897, upon the Chichester epidemic; and of Dr. C. Childs in his paper, read at this Society in the January of 1898, dealing with the historic (now happily only historic) prevalence of enteric fever in Munich, the city of Pettenkofer. I mention these only as sufficiently prominent and characteristic evidence of the recognition by the modern school of English sanitarians of the fact that too much attention has hitherto been paid to one group of factors, to the—at any rate, comparative—exclusion of others at least equally significant. Before commencing the account of our local experience of typhoid fever, I shall briefly describe, in the first place the natural soil of the city, and then the city itself.

Nottingham, Past and Present.

Nottingham stands for the most part on Bunter Sandstone (the lowest division of the Trias), of various degrees of density and hardness, and of great thickness. On the north and west, however, there is a considerable area of red marl and magnesian limestone, both of the Permian series, with some coal-crops in places. On the east again appear the Keuper marls, intersected with bands of sandstone. And, lastly, on the south, towards the Trent, and in thin and shallow lines mostly running from north to south through the town, appear respectively the alluvium and gravels of the Trent, and of its local tributaries (chief of which, of course, is the sluggish and filthy Leen).

The picturesque undulations of the town's site are due, I need hardly say, to the influence of denudation upon strata of different degress of hardness. The geological maps which I produce will show at a glance the exact extent of each formation represented, together with the surface levels (indicated by figures giving height in feet

above Ordnance datum—mean tide level at Liverpool). These levels vary from about 80 ft. at Trent Bridge to 400 ft. on Woodboro' road. So much for the natural soil and surface.

The town itself is a very ancient one. Its present name is commonly (but, I believe, erroneously) said to be derived from a Celtic word signifying "a home among the rocks," which was applied to the place in Celtic times on account of the large number of caves and underground dwellings that it then contained. The Saxon word, Snotingaham, and the Norman, Snotingham, simply meant "the home of the Snotings," and had not, as is commonly stated, the significance of "rock dwellings," though these are continually mentioned in the most ancient records of the place. The caves of the town still constitute one of its best known features, and occasionally they give rise to very serious difficulties, especially in connection with new works of building and drainage. Once, during my own time, a huge cave was found, in an ancient street in the heart of the old town known as Warser Gate, in use as a middenstead, a vast cachus common to three separate dwellings. It was stated that it had never been cleared within living memory, and we certainly experienced very great difficulty, first in clearing and cleaning it, and then in filling it up. To cut a long story short, we have the most definite evidence that a small but densely-inhabited town, having its centre to the west of but at no great distance from the great market-place, and celebrated for its cave-dwellings, has existed continuously from a remote period of antiquity. To show how circumscribed was the town's site up to comparatively recent times, I may mention that even as late as 1845, *i.e.*, before the Enclosure Act of that year came into operation, there were more than 53,000 inhabitants grouped upon such an area as I have indicated, containing only some 927 acres of ground. All this, I may say, has a distinct bearing upon my subject, for it relates to the past history of the spot upon which the city stands.

Most of you are aware that the new trunk line of the Great Central Railway passes through Nottingham, and that the Great Central and Great Northern Railways are combining to build a large central station within a few hundred yards (north-east) of the great market-place. A vast trench 13 acres in extent, about one-third of a mile long and in some places 60 ft. deep, has been dug, within which this station is now rapidly approaching completion, and many hundreds of the poorest and foulest

human rookeries in the city have disappeared from the site. While the excavations were in progress I had frequent opportunities of examining the sections of soil. The made ground above the virgin rock was in some places as much as 30 ft. deep, and contained fragments of old pottery and other relics in abundance to interest antiquarians, and enable them to gauge with considerable precision the dates of the several strata of filling. Much of the latter doubtless represented the natural accretions of successive generations of peoples and houses—such as has been described in Paris, London, and elsewhere—and much also was simple filling to level up depressions. Among the material removed were several human skeletons, and I have some bones of these now in my office from which the organic matter is by no means exhausted. Parts of the old town wall and ditch, which had not seen the light for more than a century and a half, were also cut through, and the ditch, which had been covered with a thick layer of concrete, still contained many feet of black inspissated mud cutting like cheese. I have been more than ordinarily interested in the site of the new station, by the fact that six of the men employed in the work of excavation were attacked with enteric fever during a period of eight months from the commencement of the work. Hitherto I have spoken only of the old town. The Borough Extension Act of 1877 brought the following places within the borough limits: Lenton, Basford, Radford, Bulwell, the Park, Carrington, Hyson Green, Sherwood, Brewhouse Yard, and parts of Wilford and Gedling. All these places were either country villages or small inhabited districts grouped about the town of Nottingham. All are now welded into one, not only by the Enclosure Acts, but also by more or less continuous building operations, and these latter operations are proceeding more rapidly at the present time than has been the case at any previous period of the local history. Nine hundred and forty-five houses were erected in Nottingham during 1898. The most considerable new neighbourhoods that have sprung up in recent years are those of Hyson Green, the Park, and the Meadows. The first two are built for the most part upon sandstone and loose sand, the last upon and amidst made ground—often of something more than doubtful character—above Trent gravel and alluvium. The floods of Nottingham—of which most of you will have heard—are for the most part confined to this latter district, *i.e.*, so far as the town is concerned.

The area of Nottingham is now 10,935 acres.

Water Supply.—The city is supplied with water derived from deep borings in the Bunter sandstone, all at present situated at a safe distance from centres of population. But a few words as to the past history of the local water supply. The first water company came into existence in 1700. This company had a pumping station near the foot of the Castle rock, and simply distributed the Leen water to the town. It was incorporated in 1827, and in the same year another company was formed to pump and distribute Trent water to the increasing municipality. These companies were amalgamated in 1845 (the year of the Enclosure Act). In 1856 borings in the sandstone were commenced at Basford. Fresh stations and bore-holes were established at the latter place in 1869, at Bestwood in 1872, and at Papplewick in 1882. The use of water from the Trent, the Leen, from the Trent gravel, and from rock borings within the inhabited parts of the town has long been discontinued, and only water from the above-mentioned stations, to the north of and well outside the town, is now distributed. The town council, moreover, have recently obtained Parliamentary powers to sink additional wells, in the same formation and watershed but further north (the latest station at Boughton is some 17 miles, as the crow flies, from Nottingham). The public water supply of Nottingham, then, is at the present time both pure and fairly plentiful. Twenty-one gallons per head per diem is the average amount distributed for all purposes.

System of Sewers.—The system of sewers is held—and, I believe, rightly held—to be one of the best in the country. All the sewers are of modern construction. The separate system is in use at one or two spots, but for the most part there is no attempt to differentiate the drainage. The great difficulty to be contended with has been that incidental to the drainage of land of very variable contour, with valleys of comparatively low gradient between steep hills. The valleys are now protected against risks of flood by storm sewers converging to the south-east extremity of the city, and these in times of heavy rainfall discharge direct into the Trent. At ordinary times, however, the sewage from the upper part of the town gravitates, and that from the Leen Valley and Meadows district (of which I shall have more to say later) is pumped, to trunk sewers running a distance of about 6 miles to a sewage farm (now 1,300 acres in extent) at Stoke Bardolph, on the north bank of the Trent below the town. The average daily amount of sewage reaching the farm is from 6 to 7 million

gallons. Roughly speaking, one-third of it gravitates, and two-thirds of it is pumped, to its destination.

System of Excrement Disposal.—I regret that I cannot speak in the same favourable strain of the predominant system of excrement disposal in vogue. The 40,000 pail-closets and 400 odd midden-privies, now in use, certainly constitute a very dark blot upon our municipal ménage. There are only a little over 10,000 w.c.'s of all kinds, both for domestic and business establishments, in the entire city.

Temperature and Enteric Fever.—With this somewhat lengthy introduction, I shall now proceed to give an account of the behaviour of enteric fever in Nottingham in recent years, together with some description of the conditions under which it has occurred. My first Table shows in successive years, from 1889 onwards, the typhoid cases, deaths, and death-rates, the mean annual temperatures, and the annual rainfalls. There has been a good deal of fluctuation in the annual number of cases and deaths, but it will be noticed that there was only one year in which they fell to anything like a low figure. This year was 1892; and there is a good deal that is remarkable, by way of coincidence and otherwise, about the figures for this year. The cases and deaths were alike unusually low; the numbers of male and female cases and deaths were respectively identical; the death-rate was identical with the mean rate for the great towns; and, lastly, the mean temperature of the air was no less than two degrees below the average. Of the rainfall there is not much to be said. It was somewhat below the average, but neither in distribution nor amount did it differ greatly from the falls in several other years in which the typhoid mortality was high. It is to the temperature I wish to direct special attention. If we look at the monthly records, we see that the low mean temperature was maintained throughout the year, excepting only the month of August; but even in this month it rose to only 58°, and it fell away rapidly afterwards. I have looked back over past years (prior, that is, to 1889) for records of low annual mortality from enteric fever and low temperature, and have found an almost parallel case to that of 1892 in 1879; and another, similar but not so striking, in 1888. The typhoid deaths in 1879 were only 30 in number, and that at a time too when the local prevalence of the disease was even greater than at present, and the mean temperature of the air was 44.9°. In 1888, the annual total of cases was high, because the seasonal

rise of the preceding year was prolonged till the following March; but, if we neglect the usual limits of the year, and

TABLE I.—NOTTINGHAM, 1889-1898.

General Enteric Fever Data.

Year.	1889.	1890.	1891.	1892.	1893.	1894	1895	1896	1897.	1898.
Population ...	208,826	211,695	214,606	217,550	220,551	223,584	226,659	229,775	232,935	236,139
Cases of enteric fever	367	337	375	198	479	334	422	444	428	423
Deaths from enteric fever	66	58	70	36	68	61	55	75	45	53
Death-rates from enteric fever	0.31	0.27	0.33	0.15	0.31	0.28	0.24	0.34	0.21	0.22
Mean air temperature	47.2	47.3	50.4	45.5	49.0	47.9	47.0	48.2	48.1	49.2
Rainfall in inches	25.606	17.698	25.889	21.579	20.165	20,252	20.753	22.992	23.726	19.750
Death-rates from enteric fever in great towns	0.20	0.19	0.20	0.15	0.24	0.19	0.20	0.19	0.18	0.19

examine the monthly records from March, 1888, till June, 1889, we see at once that we have to do with a period of very low comparative prevalence. In this year the highest mean temperature was recorded (as is very usual) in

August, but this highest mean was only a little over 56°, and in no other month did the mean temperature reach 55°. Although there is not, of course, a complete parallelism between seasonal diarrhœa and typhoid fever, it goes almost without saying that in these years (with deep earth temperatures scarcely touching 56°) the diarrhœa mortality was remarkably small. The actual number of deaths attributed to primary diarrhœa in these three years were: in 1879, 93; in 1888, 157; and in 1892, 158. The average annual number of deaths from diarrhœa in Nottingham during the past ten years has been something over 250.

I am aware that Mr. Hart and others have recorded the

TABLE II.—NOTTINGHAM, 1889-1898.

ENTERIC FEVER.

Cases, Deaths, and Case-Mortality, Male and Female.

—	Sex.	Age-Periods (years).			
		0—5.	5—15.	15—35.	35 and over.
Cases	Male	160	635	812	231
	Female	143	563	584	222
Deaths	Male	29	62	165	79
	Female	25	56	140	67
Case-mortality (per cent.)	Male	16.8	8.9	19.4	34.4
	Female	15.4	9.8	22.8	26.8

inhibiting influence of cold winters on water-borne outbreaks; but I am drawing special attention here to the coincidence of low-air temperature and diminished prevalence of enteric fever in connection with endemic, soil-bred disease.

Age-Incidence of Cases and Deaths.—My second Table, giving case-mortality of males and females in four age-periods, during the years 1889-98, is put in mainly with the object of increasing the somewhat scanty statistics hitherto published under this heading.

If comparison be made of my figures with those on page 26 of the Report of the Registrar-General for 1888 (dealing with 5,716 cases in the Metropolitan hospitals), it will be seen that the only at-all-noticeable difference between the two sets of figures is in the female rates in the 0-5 years and 5-15 years age-periods. In the first period my

case-mortality works out at 15.4 per cent., as compared with 12.5 of the Registrar-General; and in the second my figure is 9.8, against his 14.0. There is a very close correspondence between all the male rates in the two Tables.

Enteric Fever Incidence in Relation to Soil and Altitude.—There is not so much to be learnt as one would naturally expect from an examination of the local incidence of the disease in relation to natural soil and altitude. The bulk of our cases occur on the Bunter sandstone, and they are thickest on the poor and old neighbourhoods situated upon this formation; but, given a like amount of poverty, overcrowding, and soil-pollution, there does not seem to be much to choose (so far as liability to incidence of the fever is concerned) between residence at 100 ft., 200 ft., or even 300 ft., above Ordnance datum.

The endemic continuity of the disease also is about equally well marked among the back-to-back houses in and around Sneinton and the more open neighbourhood of Radford. Both of these are old centres of population, but the Sneinton district is distinctly urban in type, whereas Radford was originally a village with small gardens and other open spaces in the vicinity of the houses. Poverty and dirt, in combination with overcrowding, are great evils; but it does not mend matters much when the relative overcrowding of the surface is diminished, as at Radford, by the intervention of open spaces of foul, naked ground, neither cultivated nor paved. There are three areas in these districts—two at Sneinton and one at Radford—detectable at once on the spot-maps, upon which the disease is persistently endemic; and the salient features these areas possess in common are poverty, dirt, overcrowding within dwellings, unpaved or imperfectly paved ground about the latter, and the presence of the ubiquitous conservancy closet. All three are on porous sand-rock, but two are at an elevation of from 140 ft. to 200 ft. above Ordnance datum, with practically no subsoil water, while the other is only slightly elevated above high flood level.

In one dirty street at Radford (in which midden-privies predominate) the following illustrative series of cases have been recorded in consecutive years:

1895—Three cases, in May and July.
1896—Four cases, in March, August, and October.
1897—Four cases, in January, July, and September.

Four of the cases occurred in houses previously attacked.

In yet another street (where pail-closets were in a majority), there were 15 cases scattered over the 4 years

1894-97, with other four cases again recurring in the houses previously invaded.

I could cite further examples if necessary, but these will suffice, and I have chosen these specially because they illustrate at once two separate facts. The first is, the liability of the disease to hang about indefinitely in localities of this character; and the second, the comparative non-liability in such cases to anything like epidemic conflagration. There can, I think, be no doubt that long residence in an infected district, and exposure to infection, produce in many persons a considerable degree of immunity.

I have said that the bulk of the cases are on the Bunter sandstone, but so also is the greater part of the town. The wide neighbourhood known as "The Meadows," lying south of the Midland Railway line, and between this line and the Trent—to which I have already made reference—has also more than a fair share of the disease. This share, moreover, is increasing every year, and far more rapidly than the population.

The neighbourhood, as its name implies, was originally open meadow-land. Its natural soil and subsoil are Trent gravel and alluvium, overlying the Bunter, and its natural surface is on an average some seven feet above mean Trent level. The made ground rises to a height of about 7 ft. to 11 ft. above this, and its material, I know, is not always of the cleanest. On more than one occasion, in recent years, I have had to stop the use of house-refuse and privy-contents as filling for depressed ground in this district. The average thickness of the gravel and alluvium overlying the Bunter is some 21 ft., and the level of the subsoil water is practical identical with that of the Trent, so that the elevation of the roadways above the mean river and subsoil water level is from 14 ft. to 18 ft. So far as I have been able to judge, the fluctuations in the local incidence of enteric fever (in this district) have not hitherto followed those of the subsoil water in any regular manner; but what may occur later on, as the soil becomes generally polluted, I am of course unable to say.

Houses in this and other like neighbourhoods are often spoken of as being built upon made ground, but this description is not strictly accurate. I have made some little study of the subject in my own and other towns of late years, and can speak from personal knowledge of what is actually done in most cases. I will describe the exact state of matters in our own case, and in doing so shall be giving an account very fairly descriptive of a large class.

The natural surface is below high flood level, and it is necessary to raise the inhabited portions of the houses, in most cases some 7 ft. to 11 ft. above this surface, to keep them out of the water in times of flood. To do this the foundation walls are built on or in the natural soil, and carried up to a height of some 8 ft. or 12 ft. before the house proper is begun. As soon as these lower walls are completed, the roads are filled in—often with very questionable material. After this the yards and passages are also filled up to the road level. To the superficial observer all seems well. He is told that there are no cellars allowed, and, although he has only a very hazy notion of what the under parts of the houses are like, he probably rests content with this assurance. Now, the condition below the surface in such a neighbourhood is usually altogether unsatisfactory. The under parts of the houses are often far more dangerous to the health of the inmates than if they were actually used as cellars, for they are unpaved and communicate freely with the house-interiors, and yet are never inspected or cleaned, and frequently also their only possible channel of ventilation is through the houses. The roads, again, are really raised causeways above the true foundations of the houses. They contain sewers, and gas and water-mains, and any leakage from these is quite likely to find exit beneath the houses. Furthermore, the drainage from their surface and interiors naturally runs, in part at any rate, in the same direction. Now, as to the filling used in the back yards and passages. This, being less under public official supervision, is even more likely to be of questionable purity than that used for the roads. Such filling also is hardly ever properly rammed. Inequalities form in the passages and yards, drains break, and water stagnates and soaks, with the leakage from the broken drains, into the soil. This soil, moreover, being in closer contiguity to the houses than that of the roadways (the walls at the sides and rear being less substantial than in front), there is here even greater risk of pollution to the internal atmosphere of the houses than in the former case.

Hitherto I have spoken only of the state of matters underground in this district while the neighbouring river runs at its ordinary level. In times of flood, and especially of heavy flood, this state is changed, and changed very much for the worse. At such times, the water rises in soil and river; it dissolves or suspends most of the organic matter it reaches; it may even invade and wash out the sewers; and finally, after fluctuating freely in the subsoil and in the

open spaces beneath the houses, it subsides, and leaves a film of offensive mud behind.

Each house in such a district may be described as a hollow, segmented tube, with its lowest segment surrounded by foul soil, and resting upon porous alluvium and gravel subject to heavy flood. No less than 321 such houses were built in this district during 1898.

It is interesting to compare the Meadows district within the town with that lying to the south of the Trent outside the city limits, with regard to the degree of prevalence of enteric fever in each. Both are built over the same geological formation, at about the same height above the river and subsoil water, and both are equally liable to floods; but one is a sparsely-built though extensive rural suburb of good class, standing on pure soil, while the other is a dense urban district, mainly consisting of artizans' dwellings, with such unsatisfactory underground conditions as I have already described. Now, in the district south of the Trent, enteric fever has, up to the present, been of comparatively infrequent occurrence. There have been some thirty cases during the past ten years, in a population which has increased during this period from about 3,500 to 7,000; whereas, in that to the north of the river the disease, as I have already stated, is rapidly becoming endemic. The population of the Meadows district is now estimated at 20,000, and the cases of enteric fever in this district during the same period have amounted to about 350—in other words, the attack rate in the latter district has been just four times that in the former.

I regret to say that, since writing the above paragraph, I have received information from the district south of the river, which makes it appear that there also (as, but in a very much less degree than, in the Meadows) there is an apparent tendency for the disease to increase year by year more rapidly than the population. From 1889 to 1893 the cases of enteric fever numbered 11; from 1894 to 1898 they numbered 19. The simple explanation, of course, is, pollution of surface and subsoil.

Enteric Fever Incidence in Relation to Quality of Neighbourhood.—If there is one thing more noticeable than another about the regional distribution of the disease in the town, it is its general avoidance of good neighbourhoods. I do not mean to say that it never invades them, but simply that they enjoy a comparative immunity as contrasted with poorer districts. The following figures, showing the incidence of the disease during the past ten

years upon houses containing five dwelling-rooms and under, and those of larger capacity respectively, are sufficiently striking:—

NOTTINGHAM—TEN YEARS, 1889-1898.

Enteric Fever Cases.

Total Cases.	Cases in Houses of Five Rooms and under.	Cases in Houses of more than Five-room Capacity.
3.807	3,280 (86 per cent. of all)	527 (14 per cent. of all)

It is estimated that houses containing five rooms and less do not constitute more than from 45 per cent. to 50 per cent. of all houses in the city, and yet 86 per cent. of all the enteric fever cases occur in such houses

An inspection of the spot-maps at once reveals the better neighbourhoods of the town, which, as I have said, are comparatively free from enteric fever. The purely residential part known as The Park, the central business part of the town, extending roughly from The Forest to St. Mary's Church, and the eastern residential portions lying between The Forest and Sherwood Rise on the one side and Mapperly village on the other, are thus relatively exempt. To take these districts in detail. The Park is a very high-class neighbourhood, made up for the most part of large houses standing on their own cultivated grounds or gardens. Houses, grounds, and gardens are all well kept. The closets both in and outside the houses are mostly w.c.'s. The central part of the town necessarily consists of diverse elements; but the private and business houses are usually well-found and clean; their courts, alleys, and yards are well paved and drained, and they are furnished with either pail-closets or w.c.'s. Except in the Lace Market (which is mostly made up of warehouses) there is a large resident population throughout this neighbourhood. To show that the presence of our pail-closets is not necessarily productive of enteric fever, I may mention that I have a list of over a thousand houses in this neighbourhood furnished with such closets, to which no cases of enteric fever have been traced for the past two years at least.

Lastly, the residential neighbourhood lying between the Forest Recreation Ground and Mapperley may be briefly described as very similar in character to The Park; but, whereas The Park stands upon sandstone, a great part of the former is built upon clay. The comparative immunity

from enteric fever, however, is equally well marked in both.

Enteric Fever Incidence in Relation to Type of Closet.—I shall pass over the statistics of such diverse matters as the occupations of persons attacked with enteric fever, the condition with regard to paving of courts, alleys, and yards in which cases occurred, and the separation or community of curtilage of invaded houses; and proceed to discuss briefly the relation of the type of closet to degree of incidence of the fever. I have already dealt with this subject at such length and so frequently on previous occasions, that I should feel some hesitation about touching it here, were it not for its transcendent importance to many large towns, and the difficulty we still have in convincing the public of the danger to health involved in the use of dry closets in dense urban districts, and especially in their poorer neighbourhoods. The large Table I show speaks for itself. It gives the ratio of incidence of enteric fever cases, upon houses furnished, respectively, with pail-closets, midden-privies, and w.c.'s, without other distinction, except that houses and institutions with mixed closet accommodation are eliminated. I may point out that this elimination at once rids the figures of much of the fallacy attaching to the inclusion of a class of dwellings upon which the incidence of the disease is always slight—*i.e.*, good and large houses.

Furthermore, I wish to point out that the criticism which is quite admissible in the case of the comparison of incidence upon w.c. houses and dry-closet houses, without correction for neighbourhood and class of dwelling, is certainly not applicable to that having reference to the relative degree of incidence upon houses furnished respectively with midden-privies and pail-closets, for the reason that at the present time—as in other recent years—such closets when exclusively used are attached only to dwellings of the poorer sort.

During the ten years dealt with in my Table, the average annual incidence upon houses with pails was 1 in 120, upon houses with midden-privies 1 in 37, and upon houses with w.c.'s 1 in 558. That is, the proportional incidence of the disease upon houses with midden-privies was more than three times as great as that upon houses with pail-closets, and that upon houses with pail-closets more than four and a-half times greater than that upon houses with w.c.'s. If, as I just now suggested, we neglect the w.c. houses altogether, and compare only the midden-privy and

pail-closet houses, we still see that for some reason the ratio of incidence is much higher upon the first than the last, although both have dry closets. And the reason, I think, is sufficiently obvious. The high degree of pollution of air and soil almost necessarily brought about by the use of the midden-privies is the determining factor in promoting the heavier incidence.

If, now, without actually comparing the pail-closet and w.c. figures, we reflect that the first type of closet is *de facto* simply a small moveable privy aboveground, I think we shall arrive at the conclusion that the same reasons which justified the condemnation of the midden-privy apply also, though in a somewhat diminished degree, to the case of the pail-closet. And this is the more true the poorer the neighbourhood is in which the pail-closet is used. Our pail-closets, I regret to say, are mostly furnished with wooden pails. The steel pails were tried, and found less durable and less easy to manipulate—besides being more costly at the outset—hence their supercession.

"*Infectiousness*" *of Enteric Fever.*—I come now to a branch of my subject which has given rise to much controversy, and concerning which a difference of opinion exists among even the most experienced and competent clinicians. I refer to the question of the degree of practical infectiousness of enteric fever to persons dealing intimately with fever patients. This, of course, in many cases simply means the extent to which the bacillus is capable of being dried without losing its vitality. I do not intend to deal at any length with the pure bacteriology of the subject, but may remind you that, according to Germano, Uffelmann, Sidney Martin and others, the bacillus is capable of surviving the process of partial drying for a very considerable period; and only very partial drying, we know, is often quite sufficient to enable it to form a constituent of dust. I may say at once that my own mind has been long ago made up that the risk of infection to persons having at all intimate touch of typhoid patients is greatly in excess of that usually taught by the medical profession. From 1886 onwards, I have had to inquire into numerous outbreaks of the disease which, in my opinion, have been almost necessarily brought about by other agencies than the stereotyped "defective drainage," or water, milk, and foods directly infected with the poison. I may, perhaps, be allowed to make a short quotation from a report which I furnished to the Basford R. S. A. in 1887. Writing about an outbreak at the isolated hamlet of

Bleak Hall, not far from Annesley, Notts, I said: "The necessity for general antiseptic precaution I urged most strongly, being convinced that the disease was largely propagated among persons living in or visiting the hamlet, by contact with or contiguity to the sick and their infected surroundings." Again, in 1892, I was requested by the authorities at the Nottingham General Hospital to examine the drains or other possible agents of infection at that institution, with the view of accounting, if possible, for the occurence of a series of cases of the disease among members of the hospital staff. In referring to the incident in my Annual Report, I wrote as follows: "The result of my examination was entirely negative, so far, at any rate, as concerned any satisfactory explanation of the outbreak among the staff. I was driven, therefore, to the conclusion that the cases must have originated in the manipulation of the typhoid patients and their various dejections." And later I remarked: "It cannot be too fully recognised, or too earnestly taught, that there is very serious danger of infection from enteric fever patients, unless the most scrupulous personal cleanliness is observed, and the utmost care exercised to avoid the contamination of the house and general surroundings with infected matter."

At the risk of being charged with prolixity, I will venture to give in detail a few examples of the spread of the fever by what amount, clinically at any rate, to simple infection from person to person. The following extracts are from my 1893 and 1894 Annual Health Reports for Nottingham:

1893.

"Perhaps the most noticeable incidents in the local history of the disease during the year was the occurrence of two street epidemics, one at Bulwell in the summer and autumn, the other at Sneinton in the summer. The Bulwell epidemic was almost confined to Hazel Street, off Main Street; it began May 15, and ended about October 1. I could find no record of previous cases in this street. No less than 26 persons out of about 250 were attacked, and six of these died. Hazel Street proper, excluding terraces branching off on either side, and odd houses at the ends which were not invaded, is a fairly well-built street, of modern, attached, five-roomed houses. It is furnished throughout with pail-closets, clean and in good repair, and the paving and drainage of the street and yards are apparently all that could be desired. The inhabitants are mostly coal-miners, earning good wages. Notwithstanding the above facts, and the entire absence of suspicion from the food and water supply, the disease continued to hang about the street for a period, as I have stated, of four months.

"During the progress of the epidemic, the sewers and drains, and

the pavement of yards and street, were repeatedly flushed, and the closet-pails frequently changed and cleansed; the interiors of several of the houses also were disinfected and cleaned down. I directed special attention to these precautionary measures, because I was of opinion, as I still am, that the disease was communicated by direct or indirect infection of the houses, and their offices and surroundings, with the patients' discharges. In one house, No. 35, occupied by the Roberts family, the father and six children, of ages ranging from 40 to 3 years, were attacked within a period of some few weeks, and were all removed to Bagthorpe Hospital. The father had an extremely severe attack, and died in his third week; all the children recovered.

"The epidemic in Carlton Road and about Sneinton Elements was very similar in character to the above. Eighteen persons were attacked in 12 houses within a period of six months, and no common infecting agent could be discovered beyond these mentioned in the case of Hazel Street.

"Such epidemics as these, and such outbreaks as have recently occurred in our General Hospital, and others in neighbouring towns, all point to the conclusion that, in the preventive treatment of typhoid fever, it is highly important to attend with the utmost vigilance to nursing details and isolation.

1894.

"In previous reports I have given accounts of outbreaks of this disease, propagated otherwise than through food, milk, and water supplies which had been infected prior to their distribution; but I have seldom had opportunity of studying an outbreak in which there was less reason for suspecting these to have been thus previously infected than there was in one which commenced at Old Radford in September.

"George M., a coal-miner, aged 36, working at Wollaton, and living at 75 Kennington Road, Old Radford, was attacked with the disease about September 26. He was apparently infected at Wollaton, as two other men working with him at Wollaton Pit, but living outside the town, were infected about the same time. This man was laid up at Kennington Road on September 29. Prior to his attack, there had been no case of typhoid in the neighbourhood since the early part of May. George M. died of the disease on October 4. Between September 26 and December 11, eight other members of his household, his brother and brother's child, living in the same street, and occasionally visiting his house, a midwife and her daughter, also living near, who both assisted in nursing and looking after the sick household, were attacked with the disease. Later still, two other neighbours who had visited the patients became infected. The people in Kennington Road and its vicinity were repeatedly warned of the danger of infection, and how best to avoid it. Very little food appeared to be taken by the neighbours in the affected houses, and the special pails provided for the dejecta of the latter were carefully shunned; but, speaking generally, it is of course too much to expect that people of this class will be punctiliously clean and careful in their habits.

"It is extremely difficult to determine in detail the actual channels by which the infection is conveyed to each individual. I wish only to emphasise the fact that it was here, and is frequently elsewhere, conveyed in ways which, however difficult to determine in detail, are

sufficiently obvious in kind to suggest the necessity of exercising the utmost care in the treatment of typhoid fever cases among persons not yet affected with the disease. Our pail-closets are certainly not by any means entirely innocent in the matter; and, when we consider what a favourable nidus a pail-closet offers for the cultivation and dissemination of the infective poison, it seems difficult to over-rate their possible power for evil in this direction in a crowded and infected neighbourhood. Separate pails, frequently scavenged and cleaned, are provided for infected houses, but it is useless to disguise the fact that this system is more satisfactory in theory than in practice.

"I could cite numerous instances to show that multiple cases occur with much greater frequency in houses supplied with pail-closets than in those with water-closets, but the risk from pails is too obvious to call for demonstration.

"I have not space to discuss others of the many vehicles by which typhoid infection is probably carried, after its introduction from without, but I may point once more to the fact that very few of our large hospitals which admit cases of typhoid fever fail to suffer each year from the infection of members of their staffs. Our hospitals in this neighbourhood suffer continually in this way, and I have found on inquiry that the London hospitals and others in the provinces have a similar experience. A friend of mine who recently obtained a medical appointment at one of the Metropolitan Asylums Board hospitals had not been on duty there many weeks before he was laid up with typhoid fever, apparently contracted in the wards."

Hospital Accommodation.—Hospital accommodation for enteric fever cases, to the extent of some seventy beds, is at present provided between the General and Children's hospitals and the City Isolation Hospital. A block of twenty-four beds is now set apart for typhoid in the latter institution; but consent to such provision (given about two years ago) was not obtained at once, or without considerable pressure, from the Health Committee: for they persisted in regarding the disease as non-infectious, and argued therefrom that they would simply be performing the function of a charitable medical body—instead of confining themselves to their legitimate preventive work—by making such provision. I fear there is little to show in the way of diminution of cases as a result of this new departure, but it is very much otherwise with the case-mortality. The total number of cases during 1897 and 1898 were, respectively, 428 and 423, as compared with 334, 422, and 444 in 1894, '95, and '96. But, whereas the mean case-mortality for the latter years was, for males 15.4 per cent., and for females 17.1 per cent., in 1897 and 1898 it was only 11.4 per cent. for males, and 11.6 per cent. for females—thus approximating to average modern hospital mortality. The cases admitted to, or occurring in, our General Hospital during the past decennium were 870

in number, and the deaths among these 79, giving a case death-rate of only 9.08 per cent.

Although we have very little to boast of at present in the way of prevention as a result of the practice of isola-

TABLE III.

NOTTINGHAM GENERAL HOSPITAL.

Enteric Fever Cases and Deaths, 1889-1898.

Year ending with March.	Patients Admitted.	Members of Staff Attacked.	Deaths.
1889	47	3	3
1890	70	0	5
1891	82	0	6
1892	70	1	10
1893	65	7	3
1894	65	3	7
1895	59	1	7
1896	113	2	13
1897	121	1	10
1898	156	4	15
Totals	848	22	79

Case-mortality = 9.08 per cent.
Members of Staff infected per 100 patients = 2.5.

NOTTINGHAM CHILDREN'S HOSPITAL.

Enteric Fever Cases and Deaths, 1888-1898.

Year.	Patients Admitted.	Deaths.	Members of Staff Attacked.	Deaths.
1888	11	1	1	
1889	8	1	—	
1890	15	—	—	
1891	12	—	1	
1892	2	—	—	
1893	24	1	2	
1894	20	2	1	
1895	26	2	1	
1896	21	3	1	1
1897	12	—	1	
1898	6	—	3	
Totals	157	10	11	1

Case-mortality = 6.5 per cent.
Members of Staff infected per 100 patients = 7.0.

tion for enteric fever, I think we are justified in regarding it as a preventive agent of great power; especially when, as with us, hospital accommodation is provided for about

half the cases that occur, and the Health Department are entrusted with the selection of cases for removal, and with the actual removal of patients from their homes to the hospitals.

Spread of Enteric Fever through Infected Water, Milk, Shell-fish, etc.—Now, a few words with reference to the spread of the disease through the agency of infected water, milk, shell-fish, and other foods. I have, during the past decennium, recorded several small outbreaks which, in my opinion, were traceable to specifically contaminated water and milk and other ordinary foods, but I do not propose to add to the vast store of the existing literature of this subject on the present occasion. I wish only to allude briefly to the part played by shell-fish in this connection—a branch of the subject in connection with which the names of Drs. Newsholme and Bulstrode are especially well known in this country.

We have in our enteric fever register during the past two years no less than 130 recorded instance of the ingestion of such food within three weeks of the onset of fever; but at the same time I must point out that in the northern districts of Old Basford and Bulwell, where more mussels, whelks, and crabs, at any rate, are probably consumed per head of population than elsewhere—if we may judge by the enormous amount of them sold every week in these places—the incidence of typhoid fever is usually less marked than at other parts of the city of similar social class. In Basford and Bulwell the conditions under which the people live as a general rule approximate more nearly to those commonly associated with rural life than is the case in the more central parts of the town, and it may be that these conditions as protective factors (against endemic typhoid) more than counterbalance the influence of infected food-stuffs in the other direction (so far as food-borne disease is concerned).

It is not, however, strictly legitimate to compare such shell-fish as I have here mentioned upon an equal footing with oysters (*i.e.*, as regards liability to convey the living typhoid bacillus). As Dr. Bulstrode has pointed out, crabs are always, and mussels and whelks are commonly, cooked before being eaten, and the process of cooking would in most cases do away with the risk of infection. Still, a considerable quantity (of mussels particularly) would be eaten raw, and being more or less regularly and continuously taken, as I believe they are in these places, one would certainly look for more evidence of infection

through their agency than can be ordinarily found, if they were acting in this instance as regular carriers of the poison.

Summary.

I will now endeavour to summarise briefly the principal conclusions to be drawn from our local experience of enteric fever in recent years. So far as I am able to judge, they appear to be to the following effect:—

(1) That a sustained air temperature above 55° Fahr. is commonly associated as a precedent condition with an autumnal rise of enteric fever prevalence; and that a lower average temperature in the summer months is commonly followed by a comparative diminution of the disease.

(2) That the disease is endemic in thickly-inhabited and poor urban districts situated upon various geological formations (alike on the dense and the porous, the wet and the dry), and at various elevations, wherever those conditions obtain which are likely to lead to soil pollution.

(3) That good neighbourhoods, even when their dwellings are built thickly upon old made ground, and furnished with such soil-polluting agents as pail-closets, are far less liable to the endemic form of the disease than poorer neighbourhoods similarly circumstanced.

(4) That the incidence of endemic enteric fever is far heavier upon houses furnished with dry closets than upon those with w.c.'s, and upon midden-privy than pail-closet houses; and that this heavier incidence upon houses with dry closets is very much more marked in poor than well-to-do neighbourhoods.

(5) That enteric fever infection is liable to spread from person to person even in well-managed hospitals, and therefore very much more so in the dwellings of the poor.

The moral of all this, for towns like Nottingham, may, I think, be briefly stated as follows, viz.:—

(*a*) That the soil beneath the houses, and over all roadways, courts, alleys and yards in their vicinity should be sealed with impermeable and cleanable material (like Portland cement concrete).

(*b*) That all refuse should be frequently and completely scavenged, and that the curtilage of all poor houses should be periodically flushed and cleansed with water—if necessary, at the public expense.

(*c*) That water-carriage should be substituted for all conservancy methods of excrement disposal.

(*d*) That hospital accommodation should be provided for all cases of enteric fever which cannot be properly isolated or nursed at home.

NOTE.—Water-supply and drainage find no mention in this summary, because their condition at the present time in Nottingham calls for so little adverse criticism.

BLACKWATER FEVER.

By W. H. CROSSE,
Consulting Medical Officer, The Royal Niger Company, Chartered and Limited.

(*Read: March 17th*, 1899.)

Mr. President,—In venturing this evening to bring before your notice the disease called blackwater fever or hæmoglobinuric fever, I would submit that we are dealing with a subject of vital importance as regards our colonies.

It is admitted that this complaint is the cause of more deaths and invaliding than all the other diseases from which Europeans suffer in West Africa, and the same remark applies to the opposite side of the continent.

Koch, for instance, states that this disease creates the greatest havoc amongst Europeans in German East Africa.*

The disease, however, is not confined only to Europeans, for on the Congo railway many of the Chinese labourers died from it.

It has been considered to be rare amongst Africans; however, I am told that many died from it while working on the Panama Canal; and recently, we are informed,† there has been a serious outbreak of it among the blacks in the Cameroons, the disease specially attacking those who had come to the coast from the interior.

When I first went to West Africa, in 1886, blackwater fever was a complaint about which very little had been written, and the ordinary English text-books did not even mention the existence of such a disease. Dr. C. H. Eyles had published a brochure on the fevers of the Gold Coast,‡ in which a considerable amount of information is given about the disease, and an interesting account of a case of

* Koch (R.) *Das Schwarzwasserfieber. Arbeiten aus dem Kaiserlichen Gesundheitsamte*, 1898. xiv, 304.

† Plehn (F.) *Die Kamerun-Küste. Studien zur Klimatologie, Physiologie und Pathologie in den Tropen*, 1898. (Preface, p. iii.)

‡ *Malarial Fevers as met with on the Gold Coast.*

blackwater fever was published by Dr. Easmon in the *Medical Times*, August 29th, 1885, p. 277.

In 1892 I published a small book on the malarial fevers of the Niger, of which a large portion was devoted to this subject.*

The study of this complaint received a considerable impetus when Dr. Patrick Manson, on March 15th, 1893, read before this Society an interesting paper on African hæmoglobinuric fever.†

Since that time more and more attention has been given to the disease; and more especially quite recently, on account of the statement made by Koch, that blackwater fever is due to quinine poisoning.‡

I have already attempted to answer Koch's contention that blackwater fever is simply quinine poisoning, in a paper read before the Guy's Hospital Physical Society in October last.§

I have also dealt to some extent with the pathology of the disease, as evidenced by a case which I saw in England with Dr. Wooldridge, of Camberley, in a paper which I read with Mr. Pakes before the Pathological Society on December 20th, 1898.

I hope to-night to review the subject, and to lay before you a definite statement as to the opinion which I hold with regard to its pathology, causation, and treatment.

I cannot let this occasion pass without mentioning how deeply I am indebted to Dr. Washbourn, who, years ago, urged me to write on this subject, and showed me the first malarial parasites I had seen.

I also take this opportunity of thanking Dr. Howard, who has just started for East Africa, to whom I am indebted for much help in this paper. On account of his special training, we may look to him in the near future for valuable information about the disease under consideration.

Definition.

By blackwater fever, or hæmoglobinuric fever, I mean a febrile disease of malarial type, characterised by hæmoglobinuria, jaundice, and vomiting.

* *Notes on the Malarial Fevers met with on the River Niger*, by W. H. Crosse.

† *Transactions of the Epidemiological Society of London*, vol. xii, 1892-3, p. 111.

‡ Koch (R.) *Das Schwarzwasserfieber. Arbieten aus dem Kaiserlichen Gesundheitsamte*, 1898. xiv, 307-8.

§ *Guy's Hospital Gazette*, October 8th, 1898.

The name, blackwater fever, was given on account of darkness of the urine, which could scarcely fail to attract the attention of the sufferers.

The colour of the urine is due to the presence of hæmoglobin or methæmoglobin and not of blood; although in some attacks nephritis may occur, and real hæmaturia result.

Blackwater fever should not be used to include those attacks of malaria in which nephritis is a primary symptom, and which are attributed to plugging of the renal capillaries by malarial parasites.

My clinical descriptions will be based on my observations of the fever on the West Coast of Africa during nine years' service in the Royal Niger Company, in which time I saw about 100 cases.

Distribution.

Since attention has been drawn to blackwater fever, the disease has been recognised in several parts where it was formerly considered to be unknown, or, at any rate, extremely rare. For instance, when I had one of my earliest attacks on the Niger, the medical man who attended me did not recognise my illness as blackwater fever, and mistook the colour of the urine for that due to bile.

Sometimes when hæmoglobin and albumen were recognised, they were thought to be due to a renal hæmorrhage complicating the malarial attack, and up till quite recently the disease was often spoken of as hæmaturic fever. Many cases have been called yellow fever.

I do not think the disease is a new one.

Manson states that it was first described by naval surgeons stationed at Nossi Bé, a French settlement off the North West coast of Madagascar.*

Plehn quotes Bérenger-Féraud as stating that the disease existed in West Africa in 1820. He further says that it can first be proved to have become prevalent about 1850.†

The first fatal case on the Gold Coast is believed to have taken place in 1832.

It is common throughout West Africa, including the Gold Coast, Sierra Leone, Nigeria, Cameroons, and the Congo and Gaboon regions; it is also common in East Africa: for

* *Tropical Diseases*, p. 61.

† Plehn (F.) *Die Kamerun-Küste. Studien zur Klimatologie, Physiologie und Pathologie in den Tropen*, 1898, p. 104.

example, in Uganda, German East Africa, and in the Zambesi districts.

I have seen one patient who seems to have suffered from the disease in Somaliland. It has been considered very rare in India, but it is probable that it has been overlooked; for recently eleven cases have been described by Mr. Arthur Powell as occurring in Assam, and which he states were "identical with the fevers I have seen in those returned from the Gold Coast."*

It is frequent in certain tropical parts of North and South America, and in Cuba. The disease is said by Manson to have been epidemic among the labourers employed in making the canal through the Isthmus of Corinth.

Cases are reported from China, and Plehn states that its existence has also been proved in Italy, and Sicily, New Guinea, and Java.

The disease is stated to be becoming more prevalent in Africa. This cannot be entirely accounted for by the increase in the white population. I attribute its increase in certain parts of West Africa partly to the fact that, as the country is becoming more opened up, the traders live more on shore than they formerly did; for in the old days trade was in parts of the Niger Delta carried on, on the hulks which were stationed near the mouths of the river for that purpose. Another reason appears to me to be the fact that stores, houses, factories and wharves have to be erected, which of course necessitates considerable disturbance of the soil; in addition to this, large plantations are being formed, now that it is recognised that West Africa is, amongst other things, a capital coffee-growing country.

It may not be out of place here to quote from my own lecture (*Guy's Hospital Gazette*, October, 1898) the following:—

"Some will ask: Why has blackwater fever become so common recently? The first case on record in the Niger Territories is, as far as I know, my own, some ten years ago. The first case in the Niger Delta is said by some old coasters to have occurred in 1882.

"It seems to me that since we have begun to turn up virgin soil for coffee and other plantations, that the disease has become common. When I went to the Niger in 1886, there was not a single plantation. Very soon large plantations were made. I believe I was the first to start them, for I took out with me a great many seeds of Eng-

* *Journal of Tropical Medicine*, vol. i, No. 5, p. 117.

lish fruit; and soon after these were planted, an experimental plantation was made, first at Asaba, then at Abutshi, some 150 miles up the river.

"It is significant that our first three gardeners died from blackwater fever, and that for some considerable time cases only occurred near the plantations; and as plantations became more common, so the disease spread to the other stations in the territories."

Outbreaks of blackwater fever have been specially noted when the dry season has been prolonged and very hot, and when, therefore, lagoons and ponds which have been long covered with water have been dried up; the same thing has been observed when the rainy season has been broken up by long spells of drought.

Easmon, in 1881, had four cases of blackwater fever under observation, and attributes these attacks in some degree to the extreme drought of the season, drying up the extensive Quettah lagoon which had been covered with with water for twelve years.*

Climate and Conditions of Life.

The river Niger, near the sea, forms an enormous delta, and opens by many mouths into the Gulf of Guinea; this delta, which consists of mud and sand, is covered with mangrove forest and jungle; it is divided and subdivided in every direction by creeks, and, as the tide goes out, the shore shows itself as a foul mass of mud through which pour turbid streams on their way to the sea.

The level of the land is so slightly above that of the water that it is being constantly washed away, and where Europeans settle the ground is protected by pegging it round with sticks, and constantly backing them up with heavy clay, in order to prevent the sea from washing it away; to act as a further protection, old hulks and lighters are run close to the shore, and left there till they sink or fall to pieces.

The earth is constantly damp, for the subsoil water is close below its surface, and the ground is itself frequently drenched with heavy rains. The average temperature is about 80 deg. F., and the difference in 24 hours is often less than 10 degrees; the air is highly saturated with moisture, for the rainfall is very heavy, being between 125 and

* Easmon (J. Farrell). *The Nature and Treatment of Blackwater Fever*, 1884. p. 2.

150 ins. in the year. Sometimes in one day it is enormous: for instance, on turning to one of the meteorological reports of the Royal Niger Company, kindly lent to me by Sir George Goldie, the Governor, I find that at Akassa (the port of the Niger) on May 22, 1887, it was 4.080 ins.; on May 27, 1888, it was 5.905 ins.

Owing to the great amount of moisture, a mist frequently hangs over the land like a pall; whilst keys rust in one's pocket, and boots, put away for a day or two, become covered with a heavy mildew.

Periods of rain are interrupted by times of excessive drought, during which the ground splits and cracks in all directions, exhaling a peculiar stench from the decaying vegetable matter with which it is so largely impregnated. When to the above is added the fact that in the delta regions it is often difficult to obtain fresh meat, and always impossible to get a supply of fresh vegetables, it will be seen that the conditions of life are not favourable to Europeans. Finally, when we consider that mosquitoes and other blood-sucking insects abound, it will be conceded that malarial germs here find a congenial home, and in the debilitated Europeans a soil suitable for their growth; and it will not cause us surprise when we find that these regions are prolific in cases of malignant malaria.

Farther inland, say 150 miles up the river, the same conditions largely prevail; for the land, alternately drenched and scorched, is covered with a rank jungle. In the wet season the Niger is a splendid rolling river, in parts a mile broad, and capable of bearing sea-going vessels of deep draught; but in the dry season it diminishes so much that its bed is exposed in many parts, and it is with difficulty that a small vessel drawing 4 ft. or 5 ft. of water can be navigated through its narrow channels.

At the height of the wet season the land is in many parts inundated with water; native huts are flooded, and are frequently swept away. I have often seen a native fishing from a canoe in his own house. As the river falls, its banks are exposed to the fierce sun, and at low water they may be 26 ft. or 30 ft. high.

Away from the river, large ponds, lakes, and lagoons are found, and these wholly or partially dry up in the hot weather. The natives live in mud houses, under the floors of which they frequently bury their dead. To build their houses it is necessary to dig huge pits, in order to obtain the clay suitable for the purpose; these pits are not filled *up,* therefore they are found all about their towns; the

rain partially fills them with water, which stagnates, and low forms of vegetation luxuriate within them; in these pits all kinds of filth are thrown, and here snakes, mosquitoes, and other abominations find a pleasant retreat and a quiet breeding-ground.

I have said enough to show why malaria runs riot in these regions; one other factor, however, must not be omitted: I refer to the "conscientious objector"—and he is also in evidence here—for many of the Europeans have an abiding faith in the pernicious effects of quinine in all malarial cases, whether slight or severe.

I have a patient of this class now under my care. Recently he was invalided home on account of repeated attacks of malaria; his medical adviser supplied him with the quinine necessary for the voyage; but a fellow passenger knew better than the medical man, and he and some others persuaded the sufferer to take five grains of quinine every third day instead of twice a day as had been ordered. When I saw the man he was in apparently a hopeless condition, and ring-shaped parasites and crescents were plentiful in his blood. He took quinine in full doses, and in four days he was able to sit up in his front room. The poor fellow was very grateful; and as he shook hands with me, he said that he knew I had had to try the quinine kill-or-cure remedy, and he was glad I had not given him blackwater fever.

Of course it will be understood that in the higher lands of Nigeria, the conditions of life are much more favourable to Europeans.

Diagnosis and Symptoms.

Besides the hæmoglobinuria which gives the name to this disease, there are three other symptoms which are constantly present. Reports of cases are now frequent in which one or other of these symptoms is absent; such must be accepted with the greatest caution. However, it must not be forgotten that the same disease often varies in its intensity and symptoms in different parts of the world and in different patients, and that it is modified by the patient's condition of health, his food, his surroundings, and many other things.

There are, in fact, four cardinal symptoms, viz.:—

1. Fever.
2. Hæmoglobinuria.
3. Jaundice.
4. Nausea and vomiting of bile.

We will now consider these symptoms in detail.

Fever.—I contend that the hæmoglobinuria comes on during an attack of fever clinically resembling an ordinary malarial fever.

Often the temperature is not taken before the hæmoglobinuria has been observed; the patient feels, perhaps, that he has on him a more or less severe attack of fever, but does not trouble much about it, expecting that he will pull through all right as he has frequently done before.

Usually the first warning is a sharp rigor on the second or third day of the fever; the patient's temperature will rapidly rise to 103° F. or higher, and the next specimen of urine which he passes will be found to be hæmoglobinous.

Coincident with this the temperature frequently falls, often to normal; this drop I regard as probably due to the virtual hæmorrhage, and wholly analagous to the fall that accompanies hæmorrhage in typhoid fever. It has not the importance which is often given to it, and certainly does not justify the argument used by some that blackwater fever is never malarial because the temperature is not maintained for two or three days, as is often the case in ordinary African malaria. Subsequent to the drop the temperature may rise, but not as a rule very high.

In fatal cases, in which collapse is present, the temperature is often subnormal, unless it rises owing to complications. In some cases, though, there is a daily remission, yet the temperature continues to rise till death closes the scene.

Hæmoglobinuria.—The colour of the urine may be any shade between light red and that of porter, depending partly on the amount of urine passed and partly on the quantity of colouring matter present.

This is undoubtedly in most cases either oxyhæmoglobin, or methæmoglobin, or both. Thayer, however, states that the colouring matter is always present in the form of methæmoglobin.*

Albumen is, of course, always present; as a rule, bile is absent, though it is somewhat difficult to recognise in the presence of hæmoglobin; and many observers, *e.g.*, Plehn, state that they have noted it.

On shaking up the urine the froth is pink in colour. I note that Thayer mentions the greenish froth of the urine of patients suffering from hæmoglobinuric fever; if there is one thing of which I am certain, it is that the froth of

* Thayer (William Sidney). *Lectures on the Malarial Fevers*, 1898, p. 155.

the urine of blackwater fever is, in West Africa, always, as far as I have seen it, of some shade of red.

On microscopical examination none, or only very few, red discs are to be found, but hæmoglobin casts of the renal tubules are plentiful. Nothing else abnormal is constantly found; of course, as in all albuminous urine, bacteria and torulæ grow freely if it has been allowed to stand for any length of time.

Blood discs may be present after a day or so, as a result of nephritis set up by the hæmoglobinuria.

Jaundice.—It is to be noted that although jaundice is present, bile is usually absent from the urine. Copeman states that bile is either entirely absent from the urine, or is present in an insignificant amount;* at the same time there is plentiful elimination of it in the vomit and the motions.

There are none of the usual accompaniments of jaundice, such as slow pulse, itching skin, etc.

The jaundice appears to me always to precede the hæmoglobinuria. Bérenger-Féraud says that icterus appears at the outset of the attack.†

Easmon, in one of his cases (*Medical Times*, August 29th, 1885, p. 277), notes at 9 a.m. that the urine was only highly coloured, and that there was slight jaundice, and that about 3 p.m. the urine was black; and in his paper on the Nature and Treatment of Blackwater Fever (p. 2), he says that the jaundice which is uniform is seen from the very first onset of the disease.

Now we hear of cases in which jaundice appears only after the hæmoglobinuria. Up to the present, I agree with Easmon on this point; but the study of the disease is so recent, and the reported cases are so few, that I am quite willing to admit that on this, as on many other points in this interesting complaint, further knowledge may cause the older observers to make some modifications of their opinions.

The jaundice, as a rule, deepens while hæmoglobinuria is present, and gives to the patient a greenish-yellow appearance.

Nausea and Vomiting.—These are the most distressing symptoms: they are usually very persistent, and hence give much trouble in the administration of drugs and nourish-

* Allbutt's *System of Medicine*, vol. ii, p. 747.

† Bérenger-Féraud (L. J. B.), *De la fièvre bilieuse mélanurique des pays chauds comparée avec la fièvre jaune; étude clinique faite au Sénégal*, 1874, p. 118.

ment. The colour of the vomit is usually characteristic, it being of a bright or olive-green colour. Cases are reported without any mention of green vomiting; but I can only say that I have found it to be an almost constant symptom in bad attacks, and something of a guide as to the severity of the complaint; I have recorded a case (Case 12) in which there was little or no vomiting.*

This symptom is regarded as constant by Bérenger-Féraud. He says: "the vomiting is bilious, of a decided green colour; it is a constant symptom at the outset of an attack. It stains the linen of a bright green, and when collected in a basin is of a beautiful green or olive colour."†

In Easmon's case, before referred to, he states that the patient vomited a clear green fluid which contained particles like chopped spinach.

Fisher (*Guy's Hospital Gazette*, 1897) states that vomiting does not appear if the patient has an empty stomach, or if he has, some hours previously to the hæmoglobinuria, been put on a milk diet; I cannot say that this has been my experience.

Other Symptoms.—Amongst the other symptoms that go to make up an attack of blackwater fever, the following may be mentioned. Frequently there is severe headache, also pain in the loins and limbs. Patients often complain of numbness of the extremities.

Usually, there is also pain in the liver and spleen; both these organs are often enlarged.

Constipation is usual at the outset of the complaint, but later, there is danger of severe diarrhœa, especially if too active purgation has been employed; when this occurs, it will of course increase the patient's exhaustion, and cause special symptoms of its own, such as colic, straining and smarting at the anus. In one of my cases, the motions were frequent, and towards the end they became reddish in colour—due, I take it, to hæmorrhage in the bowel; melaena is said occasionally to have occurred. Often there are oppression at the chest, sighing and shallow breathing, due, I think, to the patient's anæmia.

The persistent vomiting at times causes severe gastralgia and general discomfort; and to it, no doubt, is due the excessive thirst which is so frequent. Finally, in many of the worst cases, continuous hiccough is present.

Having enumerated the symptoms of blackwater fever,

* *Notes on the Malarial Fevers met with on the River Niger*, p. 84.

† Bérenger-Féraud (L. J. B.), *De la fièvre bilieuse mélanurique des pays chauds comparée avec la fièvre jaune; étude clinique faite au Sénégal*, p. 123.

I do not think I can do better than put before you a clinical picture, describing a typical first attack as I have experienced it.

The patient, usually an adult male, is passed in England as being sound. He is sent out to Africa, and possibly goes ashore for an hour or two at Sierra Leone; some fortnight later he arrives at his destination, usually quite well, but not infrequently feeling rather heavy and bilious on account of the discomfort of the voyage, and the want of exercise on board ship; another cause of his malaise may be over-eating, or taking too much alcohol.

At first, the climate—to the new-comer—feels exhilarating, and the complete change of mode of life and surroundings is agreeable. Within a few weeks, an attack of fever supervenes; the patient may have a rigor, then a hot stage and profuse sweating. Sometimes the first attack is so severe that the sufferer loses heart, becomes useless, and has to be invalided home.

If not, he soon feels better and gets about; probably leaves off his quinine, and long before health is thoroughly re-established, returns to duty which necessitates exposure to the sun and the night air. In a few weeks he becomes ill, and is treated successfully only to fall ill again; perhaps he suffers from "bilious remittent fever," as it is called, *i.e.*, he has a severe malarial attack, with vomiting and some jaundice, and the urine contains bile.

After four or five attacks of fever, either ordinary or bilious, remittent in character, with perhaps several days when he has felt out of sorts and chilly, but has not taken his temperature, and has kept about his work, he becomes cachectic, irritable and thin, his spleen is perhaps enlarged, he is constipated, and he often feels "liverish" and out of sorts.

Later on, you get a message that he has blackwater fever. On arriving, you find him lying in bed looking pinched, jaundiced, and ill; his tongue is broad, flabby and furred, and perhaps stained green from his vomit; he complains of headache, shortness of breath, nausea, and frequent vomiting, pains in his back, and in his abdomen, especially about his liver and spleen; he is much alarmed at the colour of his urine, which in bulk looks nearly black, and which in a test-tube is seen to be dark red in colour, and when boiled is found to be loaded with albumen.

He gives the following history: he felt unwell for a few days, but did not take much notice of it, and took little or no quinine. Two days ago he felt worse—had a rigor,

perhaps—and went to bed recognising that he had a severe fever. Next day he was somewhat better, but still feverish. That night there was a row in camp, and he got up to see what it was, and felt chilled, and took some quinine which he promptly vomited. This morning he had a severe rigor, followed by a rise in temperature to about 104 F., and he noticed he was jaundiced, and that the next urine passed was deep red in colour or almost black; that his vomiting was severe, and that the vomited matter was of a decided green colour.

The patient is obviously ill, and something must be done.

He is possibly constipated, and so an enema and cholagogue purge is given. Quinine is given very freely by the mouth, for the constant vomiting makes treatment very difficult; and even when the drugs are retained, one feels doubtful whether the inflamed mucous membrane absorbs the whole of them. However, the treatment is persevered with, and with the aid of bicarbonate of soda and morphia, eventually the vomiting is relieved and some food is kept down. The temperature drops to about normal within six hours of the blackwater symptoms; on the two following days it rises to about 102 F. The urine, which never gets scanty, in the course of twenty-four or thirty-six hours clears, the jaundice which deepened at first rapidly disappears, and soon the patient gets better.

Now let me shortly describe a case ending fatally:—

The patient had been up country for four days; but feeling very unwell he came down the river in an open canoe, and was much exposed to the sun; he had a severe rigor followed by fever, jaundice, vomiting, and dark urine.

When seen he is greenish-yellow in appearance, is dulled and apathetic, the urine is scanty and dark red in colour, and rather thick, and it is passed with some difficulty.

Frequent nausea is complained of, which is only relieved for a few moments by copious vomiting. There are pain and numbness in the calves and back, the abdomen is somewhat distended; pressure over the liver and spleen—which do not appear to be greatly enlarged—gives severe pain and causes fresh vomiting.

There is considerable difficulty in breathing, and the pulse is frequent and small, and there is intense thirst.

The case gradually gets worse and worse, in spite of treatment; the urine becoming more and more scanty, till at last only a few drops can be drawn off by the catheter;

hiccough is severe, and in about a week patient dies in a comatose condition.

There are some further points connected with the clinical aspect of the question to which I would direct your attention.

Cases occurring before six months have been spent in Africa are very rare, and first attacks are uncommon—though by no means unknown—after the third year spent in Africa.

Bérenger-Féraud states that out of 100 cases of this disease in the hospitals of Senegambia, the percentage of first-year cases was 5.4; of second-year cases 22.5; of third-year cases 42.5; there being after the third year a gradual decrease.*

Hence it appears to be the rule that before an attack of hæmoglobinuria supervenes on an ordinary malarial attack, the patient must have become more or less broken down or debilitated by previous fevers. Malarial patients, who are also cachectic from other causes, *e.g.*, dysentery, are also predisposed to the disease.

I have not come across a single case in which people who have taken quinine regularly as a preventive of fever have been attacked with blackwater fever. As a rule, it will be found that the patient neglected the early stages of his attack; and there is often some special aggravating cause also to be noted, *e.g.*, exposure to the direct rays of the sun, or to chilling breezes while suffering from slight fever; but this is not always present, and occasionally a fever beginning like an ordinary one may develop blackwater symptoms without any apparent cause; in these patients there is usually marked cachexia previously.

In many of the most virulent cases the patients have resided on or near a plantation, or have been employed upon works which have necessitated excavation of the soil.

Plehn cites a fatal case—Case 7—in which the patient, a young missionary, who had lived for two years in the Cameroons, latterly suffered from several slight attacks of fever, but had never before had blackwater fever. During the last days before his attack he had been gardening a good deal; the onset of the attack the patient attributed to violent mental agitation caused by a dispute with another

* Bérenger-Féraud (L.J.B.) · *De la fièvre bilieuse mélanurique des pays chauds comparée avec la fièvre jaune; étude clinique faite au Sénegal*, 1874, p. 109 (not p. 107, as stated in *Plehn, Kamerun-Küste*, p. 106.)

missionary* (I would myself attribute the attack to the disturbance of the soil rather than to that of his mind).

A rigor usually precedes the onset of the blackwater symptoms; this is not, however, as is often stated, the true beginning of the disease; the patient has almost invariably more or less fever for at least a day or two previously.

The hæmoglobinuria usually passes off gradually in two or three days, and rarely returns during the attack. Occasionally, however, a succession of rigors occur, and the urine is only hæmoglobinous for a short time after each rigor. Davidson gives an account of such a case.†

Cause of Death. — There are two main causes of death:—

1. Suppression of the urine from secondary nephritis set up by the passage of hæmoglobin through the kidneys; the urine is scanty, and there is hæmaturia followed by suppression and uræmia; this has been put down to quinine poisoning, but the patients are often only first seen by the medical man when this condition is already established, and not infrequently before they have taken any quinine. Frequently also they are able to retain very little quinine when it is administered.

2. Exhaustion. Anuria may be present, but it is due to the collapse. In these cases the urine becomes clear, but there is profuse perspiration; the pulse becomes feeble, and the patient gradually sinks. The urine is scanty, as in all cases of collapse, and it may be almost suppressed, but it does not contain blood.

In certain cases, patients are stated to have died from shock due to the enormous destruction of the red discs.

The mortality in Nigeria due to blackwater fever appears to be about 20 per cent. of those attacked.

That the death-rate is high is hardly surprising, when we consider that in opening up a savage country like Nigeria, many men are necessarily cut off for days or weeks from their medical officers, and that proper hospitals and nursing are often absent. In addition to this, the well-known prejudice against quinine, and the folly and excesses of some men, must be taken into account.

* Plehn (F.) *Die Kamerun-Küste. Studien zur Klimatologie, Physiologie und Pathologie in den Tropen*, 1898. pp. 112, 113.

† *Hygiene and Diseases of Warm Climates*, p. 181.

Subsequent Attacks.

Two kinds must be recognised:—

I. *True Relapses.* —These may occur within about a week of the primary attack; it is well known that such relapses are liable to take place with ordinary African fevers, if the patient has got about too soon and has been inadequately treated. The same thing may occur in blackwater fever. These should not, in my opinion, be called second attacks, as they occur before the patients are well: sometimes, while they are still in bed; they are usually mild as to the amount of hæmoglobinuria, but on account of the great prostration of the patients are not without danger.

II. *True Second Attacks or Recurrences.*—A patient who has had blackwater fever is more liable to suffer from the disease than one who has not. This is especially true if he stays in tropical Africa after the attack, for he will probably remain in the cachectic condition which renders him liable to a recurrence; and even after restoration to health by a holiday in England, on his return to Africa, if he gets into the old condition of cachexia, he is very likely to suffer from a fresh attack.

Moreover, second attacks or recurrences may occur without fresh infection in a manner precisely similar to ordinary malaria; thus, such an attack may occur on the voyage home or in England. Second attacks are usually more fatal than primary, and third attacks are worse still; but in rare cases, after two or three attacks, some kind of tolerance seems to be developed.

It will now be seen that the distinction which I intend to convey by using the words "relapse" and "recurrence" is important: a relapse is usually slighter, and a recurrence more severe than the primary attack. Moreover, confusion between the two leads to fallacious statistics. Thus, a man may say that he has had four attacks of blackwater fever, three of which occurred in two weeks, and the other one and a-half years later. This I regard as only two distinct attacks, two relapses having occurred in the first blackwater fever.

Examination of the Blood.

The difficulties of obtaining reliable information are very great. A medical man is at a great disadvantage; he is frequently worn out with nursing or sickness, and has little

energy left for scientific inquiry. Owing to the difficulties of porterage, often he cannot carry a large microscope and outfit with him.

As an instance of his difficulties, I may mention that when the Brass men raided our port on the Niger in 1895, they annexed everything they could, and amongst other things, our microscope, which is now, I understand, decorating a Fetish temple; and no doubt my operating case—which was also carried off—has performed a not-unimportant part in the sacrifices which were said to take place almost every day.

Since Manson's ingenious device of spreading large films with the aid of cigarette paper, the examination of blood specimens has been rendered much more easy. The method has been elaborated by Pakes and Howard; and I strongly advise anyone interested in this subject to read the plain and definite rules for spreading and fixing and staining blood films, published by them in the *Journal of Tropical Medicine* for February 1899.

Even when the observer is competent, and his methods are good, and though he feels certain that the patient is malarious, yet in the peripheral blood it may be almost impossible, without prolonged observation, to find a single parasite.

Now it is well known that the parasites of the æstivo-autumnal group are only present in the peripheral circulation during their earlier unpigmented stage of development, and even then in comparatively small numbers; and that most of the reliable work on their morphology and development has been done in Italy, where opportunities of examining blood aspirated from the spleen, or fixed in sections of organs obtained from post-mortem examinations, are available. In Africa it is scarcely justifiable to aspirate the spleen for scientific purposes, and the number of properly worked out post-mortem examinations are few.

Still, in spite of these difficulties, parasites have been found in the peripheral blood in cases of blackwater fever, and the more constantly the more competent the observer; they resemble the æstivo-autumnal group in that, until now, only young unpigmented forms have been found in the peripheral circulation. Whether identical with one of the three varieties already described as occurring in Italy, or distinct from them, remains to be worked out, and this can only be done by finding older forms in the blood of internal organs.

F. Plehn, who has done so much good work in this

connection, has described an organism found in the blood, apparently identical with that found in ordinary malaria in Africa; and recently A. Plehn* has recognised the identity of the parasites so described with the æstivo-autumnal parasite.

Mannaberg, referring to blood examination in this disease, gives us the following warning:—"Regarding blackwater fevers in which the search for parasites proves negative, it is to be remarked that this proves little when the blood is not examined at the very beginning; less when the patient has already taken quinine."†

Manson says:—"Plasmodia have been found in the blood and organs; they are small, and rarely sporulate in the peripheral blood, belonging doubtless to some form of the crescent-forming malignant type."

In three slips which I recently took from a patient in England suffering from blackwater fever, we found nine small circular unpigmented parasites with vesicular nucleus about one-fourth the size of a red corpuscle. I think you will agree that, so far as microscopical examination of peripheral blood is concerned, they are indistinguishable from the unpigmented æstivo-autumnal parasites observed in Italy.

Pathology.—Having thus laid before you, to the best of my ability, the known facts relating to this disease, I think that we are in a position to discuss the vexed question of its true nature and epidemiology. Four main views have, from time to time, been propounded.

These views are as follows:—

1. That the disease is nothing more nor less than quinine poisoning.

2. That the disease is not malarial, but is a distinctive entity, and one which is especially liable to attack those who are debilitated from malarial and other causes.

3. As far as I can follow Sambon, he appears to believe that the disease is the same as paroxysmal hæmoglobinuria.‡

4. That the disease is a complication of malaria. This is the view which I have always advocated, and one which, I venture to think, is held by nearly all those who have had extensive clinical experience of it; especially, I would

* *Beiträge zur Kenntniss von Verlauf und Behandlung der tropischen Malaria in Kamerun.* Berlin, Hirschwald, 1896.

† *Die Malaria-Krankheiten,* 1899, p. 225.

‡ *British Medical Journal,* September 24th, 1898, p. 868.

mention Easmon, the first British subject, I believe, who described the disease.

1. Koch's theory is that the disease is not a malarial one at all, but that it is directly due to the administration of quinine; but he further says that, although no case came under his notice in which quinine poisoning could be discarded, yet that he would not go so far as to say that all cases are due to quinine poisoning.*

In answer to this, I can say confidently that I have seen cases of blackwater fever in which no quinine has been taken. Dr. Cargill has reported cases to me which bear out this statement. Recently in England I have seen a case of blackwater fever, in which the patient had taken no quinine before the blackwater symptoms set in. This case is of considerable interest, for the patient, though he had many attacks of ordinary fever in Africa, and for which he had taken large doses of quinine, never had blackwater fever till he developed it some days after he had arrived in England, in bitter weather, debilitated with fever and dysentery, and when he did not take quinine. A further point of interest is that while taking quinine by the mouth he had a slight relapse; the acid-hydrochloride of quinine was then administered in large doses hypodermically, and was continued for some days, and yet there was no further hæmoglobinuria, and the patient ultimately recovered from the subsequent anæmia as well as from his dysentery.

Again, Surgeon G. McGregor, writing to the *Guy's Hospital Gazette*, January 21st, 1899, from H.M.S. *Mosquito*, Zambesi River, after stating that he has had experience of malaria in the West Indies, in West Africa, and on the Zambesi and Shire rivers, remarks:—

"Of the so-called blackwater fever, which I regard as a pernicious form of malarial fever, I have had three cases under my care, and in all of them quinine was given in doses of 45-60 grains in twenty-four hours throughout the illness, and all recovered. And not only is quinine administered during an attack of fever, but the bluejackets here take five grains of quinine night and morning for several months at a time, and blackwater fever is rare amongst them, although they suffer from the ordinary remittent and intermittent types, but in a much less aggravated form than occurs amongst residents on shore."

Later on, he strongly asserts that bad drinking-water

* Koch (R.), *Das Schwarzwasserfieber. Arbeiten aus dem kaiserlichen Gesundheitsamte*, 1898, xiv, 308.

and the absence of regular doses of quinine account for very severe types of fever which occur on shore.

On the Niger all cases were treated with quinine, and a large percentage of these recovered; surely these cases should have got worse under quinine treatment if Koch's statement is correct.

I have personally had many attacks of the disease: in all I have taken quinine; and though in England I have taken large doses for severe fever, yet I have not again developed any blackwater symptoms. My experience is that blackwater fever comes on chiefly in those who have neglected to take quinine. I have certainly had a case in which a man who had had one attack of the disease escaped any further attacks, apparently on account of the quinine which he took regularly.

Again, many cases have been reported in which patients have recovered from the disease when heroic doses of the drug have been given; I would refer specially to the cases reported by Dr. Moffatt, Principal Medical Officer, Uganda Protectorate; out of nine cases treated two died: in both of these he says that quinine was neglected till too late; all the other cases were treated with quinine, six of them with doses of 60-120 grains in twenty-four hours (*British Medical Journal*, September 24th, 1898, p. 926).

Dr. Robson, of Birmingham, who was in West Africa for two years, writes to the *British Medical Journal*, October 22nd, 1898 (p. 1287), as follows:—

"Again, in the most severe cases of blackwater fever I have seen patients get well who have been heroically dosed. I have myself, on more than one occasion, administered 60 grains (of quinine) as a single dose with advantage. . . . . When the tropical physician has a better remedy supplied him, no doubt he will relinquish his old favourite, but assuredly not till then."

Battersby, who suffered from more than one attack on the Niger, regards this complaint as purely malarial.

Eyles gives his opinion of the disease in the following words:—

"In short, this is the view I take of blackwater fever that it is a malarial remittent fever, to which is added another malarial manifestation, viz., hæmoglobinuria; and that when this occurs there is more marked hepatic disturbance than in ordinary remittents.*

I have elsewhere referred to a case of hæmoglobinuric

* *Malarial Fevers as met with on the Gold Coast*, p. 11.

fever cited by Davidson in his book, *Hygiene and Diseases of Warm Climates* (p. 181), in which the disease was not cured till quinine had been given very freely.

I think that these quotations (and I could give many others) suffice to prove that in most cases blackwater fever is not quinine poisoning. Is it so in any cases? Cases have been recorded, chiefly in Italy and Greece, where, in certain patients, owing to some inherent susceptibility, even minute doses of quinine were able to excite an attack of hæmoglobinuria; and whenever a subsequent dose was given, whether during an attack of malaria or not, another attack took place. It is said that sometimes this has been observed in several members of the same family. It is to be noted, however, that these results are very few in number, and have only occurred where the patients lived in malarial districts and suffered from malarial cachexia; and that some have previously had blackwater fever apart from quinine, and that the result is apparently independent of the amount of quinine given, a very small dose sufficing. Also, it is to be remembered that hæmoglobinuria is never a symptom of quinine poisoning in non-malarious subjects.

These cases must be taken for what they are worth. It is possible that very occasionally quinine may cause hæmoglobinuria, but this is an idiosyncrasy, and cannot be regarded as the cause of blackwater fever in Africa, for no one would be found to assert that the African cases infallibly relapse every time a small dose of quinine is administered, as is the case with these.

2. The second theory is that blackwater fever is a distinctive entity. This view was put forward by Manson in his able paper,* the main arguments being based on the distribution of the disease. Since then, fresh facts have been brought to light; and in his recent work on tropical diseases, towards the end of his article on hæmoglobinuric fever, he speaks of it as being "the most important of the African forms of malarial poisoning." It has been pointed out that the distribution and relative prevalence of blackwater fever do not correspond with that of the æstivo-autumnal parasite; and that in some regions where the latter was very prevalent, blackwater fever was very rare or almost unknown. In answer to this I would urge two points: in the first place, since attention was drawn to the disease it has been described in regions where it was thought not to exist; thus, Dr. Arthur Powell writing

* *Transactions of the Epidemiological Society*, 1892-93, xii, p. 111.

on hæmoglobinuric fever in Assam, says: "The existence of this fever in India was denied some years ago, but whether justly so or not, it is now either undoubtedly on the increase or, perhaps, more accurately diagnosed, where formerly it was confounded with bilious remittents." After noting that Crombie says it is practically unknown in India, he goes on to say: "I now record eleven cases which occurred in this district, which were identical with the fever I have seen in those returned from the Gold Coast."*

Secondly, according to the Italian observers, æstivo-autumnal fever is by no means the entity which it is often assumed to be. No less than three distinct parasites are described, viz: malignant tertian, malignant pigmented quotidian, and malignant unpigmented quotidian. It is very possible that there may be one or more other varieties which occur in tropical Africa and which have a distribution similar to blackwater fever, and with which blackwater fever is associated. Evidence in support of this is scanty, but it will be remembered that the three kinds of Italian malignant parasites have been only quite recently differentiated; indeed, the conclusions of Italian observers are still *sub judice*, and the question can only be settled by careful examination of the blood in internal organs, where alone the later development of the malignant parasite occurs.

3. Sambon's view that blackwater fever is paroxysmal hæmoglobinuria is sufficiently demolished by the fact that parasites are found in the blood of blackwater fever.

4. We now come to the fourth alternative, viz., that blackwater fever is not a distinctive disease, but is a complication occurring in the course of malaria. I venture to think that there is a large amount of evidence in favour of this view. It is certainly the view held by most African practitioners; I will endeavour to prove that it is a reasonable and natural hypothesis. In the first place, I would assert that the one predisposing cause present in almost all cases, is that the patients are previously suffering from a pronounced malarial cachexia; this was very clearly brought out in a series of cases recorded by Dr. Arthur Powell in Assam; it is further borne out by my own nine years' experience, and by the statements of all the other West African medical men with whom I have been brought in contact; for instance, Mr. J. J. G. Whittindale, Dr. F. Cargill, and my successor, Mr. E. E. Craster.

* *The Journal of Tropical Medicine*, December 1898, p. 117.

I cannot agree with Dr. Sambon's statement, "We know of several cases now in which melanuric fever was contracted within a few weeks of landing and without any previous attack of malaria,"* and I would draw attention to the fact that this statement is apparently contradicted in another part of the paper, for I find it stated that "It is of the highest importance to note that blackwater fever is seldom a primary disease. Authors, from all parts, are unanimous in stating that the liability to infection is constantly associated with the occurrence of malaria."†

Dr. Easmon says: "It (blackwater fever) only attacks those whose healths have, for some reason or other, become deteriorated." Further, he says, "It is often preceded by simple attacks of intermittent fever, with progressive anæmia and sallowness of the countenance."‡

Charts 6-9 in my book show that between December 5th and January 30th four malarial attacks—one of which was decidedly remittent in type—preceded an attack of blackwater fever which was intermittent in character.

As further evidence, I may mention one case which came under my notice, of a new-comer who developed blackwater fever within a few months of his landing in West Africa; but in this case it was found that the patient had previously suffered from severe malaria in Burma.

This condition of cachexia may be brought about either by several definite attacks of severe malaria, or perhaps by frequent slight attacks which are at times not even severe enough to keep the patient in bed, and which are therefore neglected. In the latter case, though the patient feels a little out of sorts and chilly, he usually disregards it, and does not take his temperature; or he may take it and find it to be 100° F., swallow some antipyrin or quinine and go to bed, and then get up next day feeling rather better, and go about his work. I take it that though such a patient would tell you that he had been in good health and had had no fever, yet malarial parasites had been present in his blood the whole time; and that though his power of resistance was just sufficient to keep them from multiplying to any great extent, still, all this time they were manufacturing toxines, and thus contributing to that toxæmia to which malarial cachexia is due.

Such persons as these are the subjects who are most liable

* *British Medical Journal*, September 24th, 1898, p. 868.

† *British Medical Journal*, September 24th, 1898, p. 867.

‡ Easmon (J. Farrell), *The Nature and Treatment of Blackwater Fever*, 1884, p. 2.

to suffer from blackwater fever, and I will go further and say that I am certain that when it arises in such cachectics its occurrence is due either to excessive exposure, privation, great fatigue, to some special malignancy of the parasite, or to some indiscretion committed by the patient during an attack of ordinary malaria which might otherwise have passed off without this complication.

Cause of Hæmoglobinuria.—Before discussing the cause of the hæmoglobinuria in blackwater fever we must look at the process by which destruction of hæmoglobin in malaria ordinarily takes place. In benign tertian and quartan fevers the parasite destroys the hæmoglobin and turns it into another pigment, and when it sporulates this pigment is set free and carried to liver, spleen and brain, causing the well-known malarial pigmentation. In the æstivo-autumnal fevers the same thing occurs, but the parasites cause the rupture of the corpuscles before the hæmoglobin is completely destroyed, thus causing hæmoglobinæmia. In ordinary fevers this free hæmoglobin is taken up by the liver and changed into bile pigment, but none appears in the urine. In severe æstivo-autumnal fevers one-third of the total number of corpuscles may be destroyed in one paroxysm and yet no hæmoglobinuria occur, the result of the excessive bile formation being bilious remittent fever.

For hæmoglobinuria to occur there must be a large number of corpuscles destroyed and a large quantity of hæmoglobin set free in the blood; this is proved by the experimental hæmoglobinuria induced by certain poisons. Hence, in order to explain the occurrence of hæmoglobinuria in malaria we are compelled to assume that there is a sudden destruction of corpuscles, thus setting free the whole of their hæmoglobin. Evidence in favour of this is to be found in some of the descriptions of the result of examination of fresh blood films during an attack of hæmoglobinuria. Thus Manson speaks of the apparent ruin and wholesale solution of the red discs. What change can it be that brings about this destruction? A. Plehn suggested that it was some alteration in the specific gravity of the blood, causing solution of the red discs. I would rather suggest it was due to the accumulation of malarial toxines. As above stated, the patients are cachectic, *i.e.*, they are suffering from chronic toxæmia. Supposing anything, *e.g.*, chill, should check the excretion of toxines, they might accumulate until they were able to effect the direct destruction of the red discs. On this theory, blackwater fever is to be regarded as a special complication of malaria.

The hæmoglobinuric complication I regard as wholly analogous to chlorate of potash poisoning, and paroxysmal hæmoglobinuria. In blackwater fever the cause is probably the malarial toxine; in chlorate of potash poisoning it is sufficiently obvious; in paroxysmal hæmoglobinuria it is unknown, though it possibly is syphilitic in origin, as suggested by Copeman,* who says: "A definite specific history has been forthcoming in all the cases that have come under my own observation." The three cases are, however, only analogous. I do not for one moment suggest the identity of the causes of blackwater fever and paroxysmal hæmoglobinuria, any more than I should of blackwater fever and chlorate of potash poisoning. The hæmoglobinuria is simply the natural result of the preceding hæmoglobinæmia.

One other fact certainly is of importance in blackwater fever, as in all other poisoning cases, and that is idiosyncrasy. I believe that some persons get hæmoglobinuria with a much lower degree of malarial toxæmia than others. Some of these patients may account for the slight and frequently recurrent cases that are reported.

Another factor may come in, and that is degenerative changes in the liver, the result of malarial poisoning, which impair its bile-forming functions. It is clear that if the liver were to strike work, the hæmoglobin would not be changed into bile pigment, and that a hæmoglobinæmia, however slight, would cause hæmoglobinuria. That such, however, is not the main cause, is, I think, shown by the fact that there is much bile formed during blackwater fever, as shown by the profuse bilious vomiting and the not-infrequent bilious diarrhœa.

While not wishing to claim that the above is any more than a possible theory as to the cause of blackwater fever, yet I would submit that it is possible, and further, that it helps to emphasise what I believe to be undoubted facts—viz.: that blackwater fever is of malarial origin, and further, that it is a special complication occurring in malarial cachectics, and dependent on the destruction, by some means other than direct parasitic invasion, of the red discs and the solution of the contained hæmoglobin in the plasma.

* Allbutt's *System of Medicine*, vol. v, p. 627.

TREATMENT.

Having thus discussed the pathology of blackwater fever, and shown what I hold to be its connection with malaria, I come to the practical question of its treatment. In dealing with this, it will be well to remind ourselves that three distinct factors are present.

There is, in the first place, the attack of malaria during which the hæmoglobinuric paroxysm develops.

Secondly, there is the chronic malarial toxæmia which has predisposed the patient to the attack.

Thirdly, there is the sudden loss of hæmoglobin, which in its effect in no way differs from an ordinary severe hæmorrhage.

The above are present in all cases; sometimes there is another factor present in the subsequent nephritis.

It is clear that only the first of these, namely, the malaria, would yield to quinine. So far as is at present known, quinine has only a parasiticide and not an antitoxic function in malaria; and hence it cannot be relied upon for treatment of toxæmia, and obviously not for the hæmorrhage.

Quinine, therefore, should be given in blackwater fever, but with the express object of clearing the blood of the malarial parasites, and hence it should be given in full doses similar to those given in ordinary attacks of tropical malaria.

It must, however, be clearly understood that by the time a patient has reached the point of hæmoglobinuria, his tissues are probably much degenerated and his vital functions seriously lowered; hence, though we may cure his malaria by quinine, we must not be surprised should he succumb.

A cachectic patient may be unable to withstand a severe hæmorrhage; when to this is added the constant and distressing vomiting which often involves starvation, he is clearly liable to die from exhaustion, as many of the cases certainly do.

On the other hand, this is a very different thing from saying that quinine is of no use in blackwater fever. I hold that it should be always administered in doses sufficient to make it certain that the blood is freed from parasites: just as the first step in the treatment of a patient suffering from sapræmia due to retained placenta is to clear out the uterus, and so remove any danger of further infection.

As previously mentioned in this paper, it is frequently

difficult to efficiently administer quinine by the mouth on account of the persistent vomiting, and administration by the rectum is unsatisfactory. I am, myself, a strong advocate of the hypodermic administration of quinine under these circumstances; and in this practice I am supported by the profession in Italy and America—though, as far as I know, many English medical men hesitate to adopt it. The advantage of the hypodermic method is sufficiently obvious, for one can rest assured that the dose which we administer is actually absorbed.

The supposed difficulties are really hardly worth our consideration if we are thoroughly aseptic.

Sulphate of quinine is not suitable for hypodermic injection; perhaps the best salt is the acid-hydrochloride which is chiefly used in Italy: it is soluble in its own weight of water. The acid-hydrobromide which is soluble 1 in 6 of water may also be used, especially if the patient is liable to cinchonism.

Personally, when in Africa I used the lactate which is soluble (according to Squire, Seventeenth edition, 1899), 1 in 6 of water. I am inclined to think that 10 grains of quinine administered hypodermically every eight hours on the first and second day of the disease, and every twelve hours on the third day, would be a sufficient dose; after this, I suggest that five grains should be given twice a day till the end of the attack. I may mention that I always boil the solution of quinine, and inject it while it is still quite warm. When the drug cannot be administered hypodermically, larger amounts may be necessary.

In a case of blackwater fever which I recently saw in England, quinine was at first given by the mouth; the patient had a relapse; it was then given hypodermically, and the patient recovered without any further relapse.*

It is, of course, to be remembered that quinine will accumulate if the urine is either greatly reduced or suppressed, and therefore it must not be continued in the same doses should this contingency occur.

So much for the treatment of the malarial element; as regards the other two factors, the main indications are to maintain free excretion, with the view to liberating the body from the accumulated toxines, and to procure rest and nutrition which will enable recovery to take place from the anæmia.

If the bowels are not opened, a cholagogue purgative and

* Wooldridge (A. T.). A case of Blackwater fever complicated by dysentery. —*Lancet*, March 18th, 1899, p. 762.

enema should be given; but over-purgation should be avoided. I always myself administer sodium bicarbonate freely: this helps to allay vomiting and gastric pain, and makes the patient more comfortable; and, I think, does good by its diuretic action, and also helps to diminish the acidity of the urine.

Opium should be given without hesitation to procure rest. Nutrition must be maintained by rectal feeding if necessary.

Strychnine and digitalis may be necessary in cases of extreme asthenia; they will act beneficially by helping to maintain the renal blood pressure, and therefore the secretion of urine: for, doubtless, some of the cases of anuria occurring in blackwater fever are the result of collapse and not of nephritis.

If nephritis occurs, the usual remedies are to be adopted, but caution should be used in the administration of such depressing drugs as pilocarpine, from the use of which I have certainly seen disastrous results; if it is decided to administer this drug, not more than one-sixth of a grain should be given at one time.

After recovery, the patient ought to be advised to take a holiday, if possible, in England; and should not return, at any rate, until he has completely recovered from his anæmia and cachexia. Indeed, on general principles it is probably desirable that no one should return to Africa after an attack. This is particularly the case where the blackwater fever has developed in spite of precautions, but applies less to those cases where the attack can be traced to exceptional hardship and privations, which are not likely to be repeated.

Those who, from one cause or another, cannot stand quinine, and those who suffer from even slight albuminuria, should not be selected for malarious climates.

Finally, I urge that the prophylaxis of blackwater fever by habitual use of quinine will be found to be more hopeful than its treatment.

I have come to the definite conclusion that, besides adopting precautions in regard to exposure to chills, wet, and to the sun, and as to moderation in food, alcohol, and sexual matters, every resident in the more malarious parts of Africa should take at least five grains of quinine each day.

In the large majority of cases I believe that this would prevent the chronic malarial poisoning which is the predisposing cause of blackwater fever; and would do much to eliminate this scourge from our colonies.

History of Case.

A. B., 26 years of age, had resided for 14 months in Nigeria, and during that time he had had many attacks of malarial fever and three of dysentery; he also suffered from fever on the voyage home. On his arrival, on November 25th, he was suffering from dysentery and had taken a severe chill.

November 26th, went to bed; took no quinine till the morning of the 27th, when he swallowed and promptly vomited a five-grain tabloid. He had a severe rigor on the 27th, at 11 A.M., and the urine became hæmoglobinous at noon on the same day. There was a slighter rigor on the 28th. Jaundice and green vomiting were marked on the 27th, 28th and 29th.

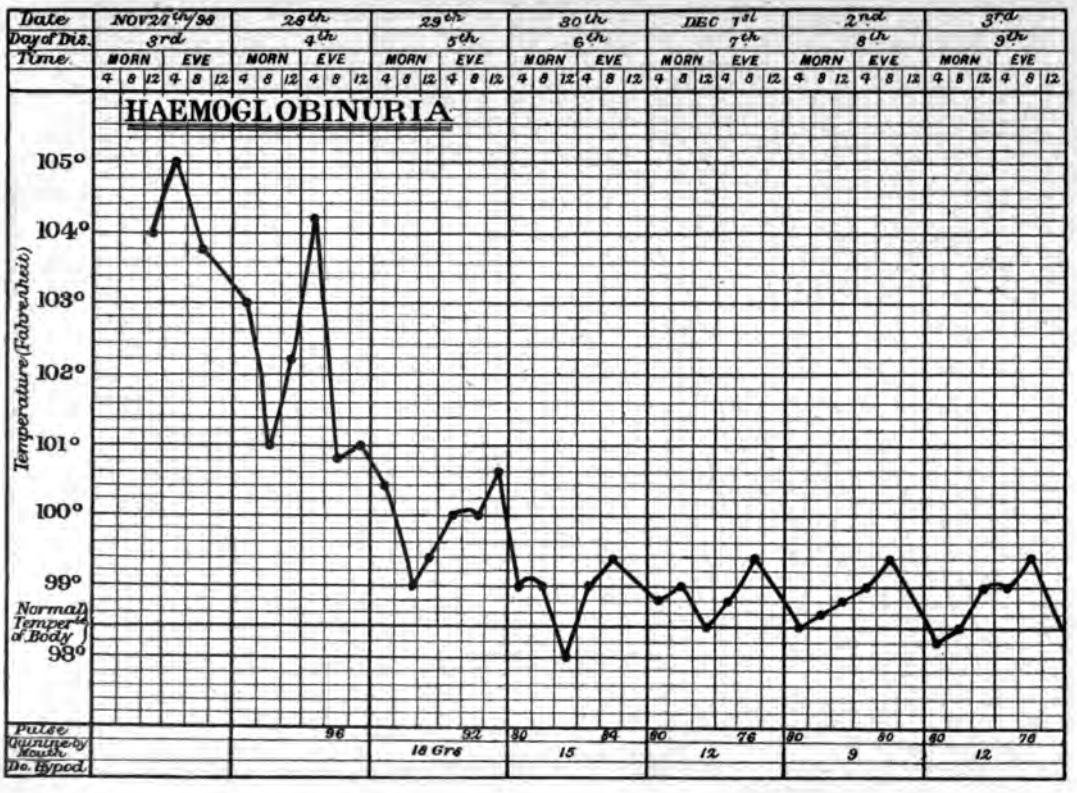
December 4th, patient became more sallow in appearance. December 5th, the urine passed between 4 P.M and 8 P.M. was hæmoglobinous.

December 8th, as the temperature was rising, quinine was given hypodermically and by the mouth. After this there was no more hæmoglobinuria, and the patient gradually convalesced, his recovery being retarded by his dysentery and anæmia, and by a slough caused by one of the hypodermic injections of quinine, this being the only one out of fourteen which caused any trouble. There was no tendency to suppression of the urine except on November 29th, when only sixteen ounces were passed; bicarbonate of soda was then administered, and thirty-three ounces of urine were passed on the next day.

Quinine taken before the attack	...	None.
Quinine taken to end of December 5th,	hypodermically ...	None.
" " "	by the mouth ...	111 grs.
Quinine taken from Dec. 6th to 15th,	by the mouth ...	117 "
" " "	hypodermically ...	102 "

Total—by mouth 228 grs.; hypodermically, 102 grs. = 330 grs.

In this case it cannot be said that quinine caused the attack, nor is it likely that it had anything to do with the slight relapse.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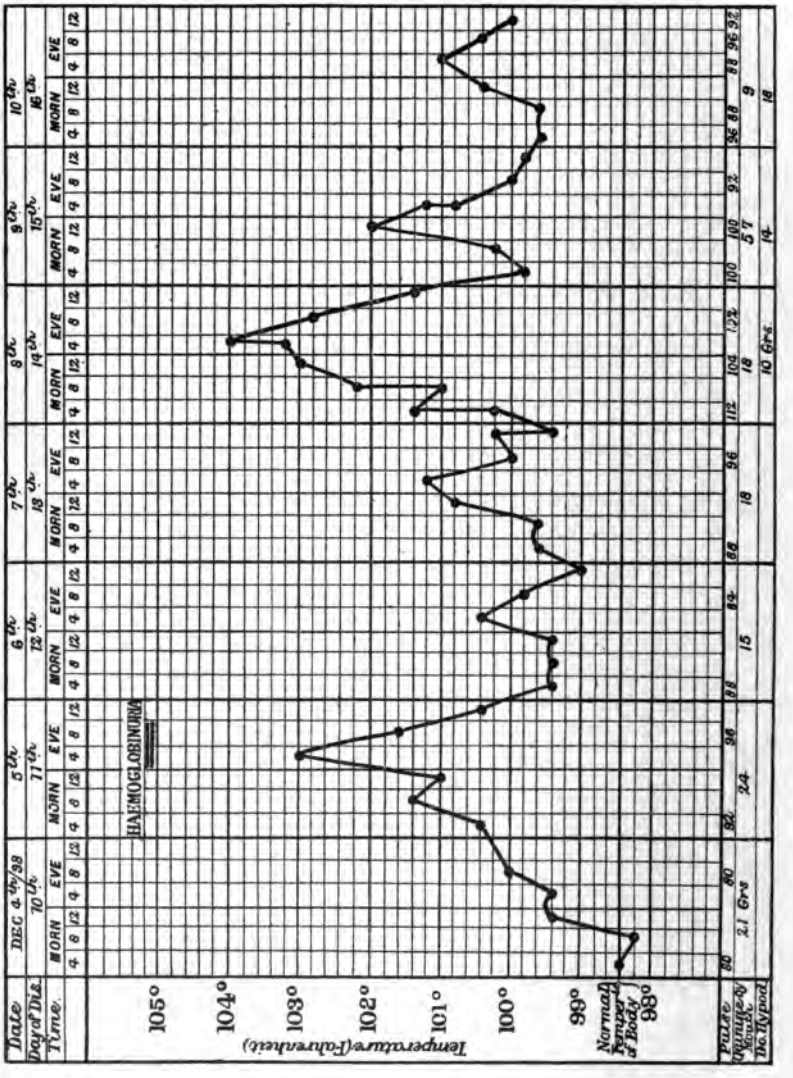
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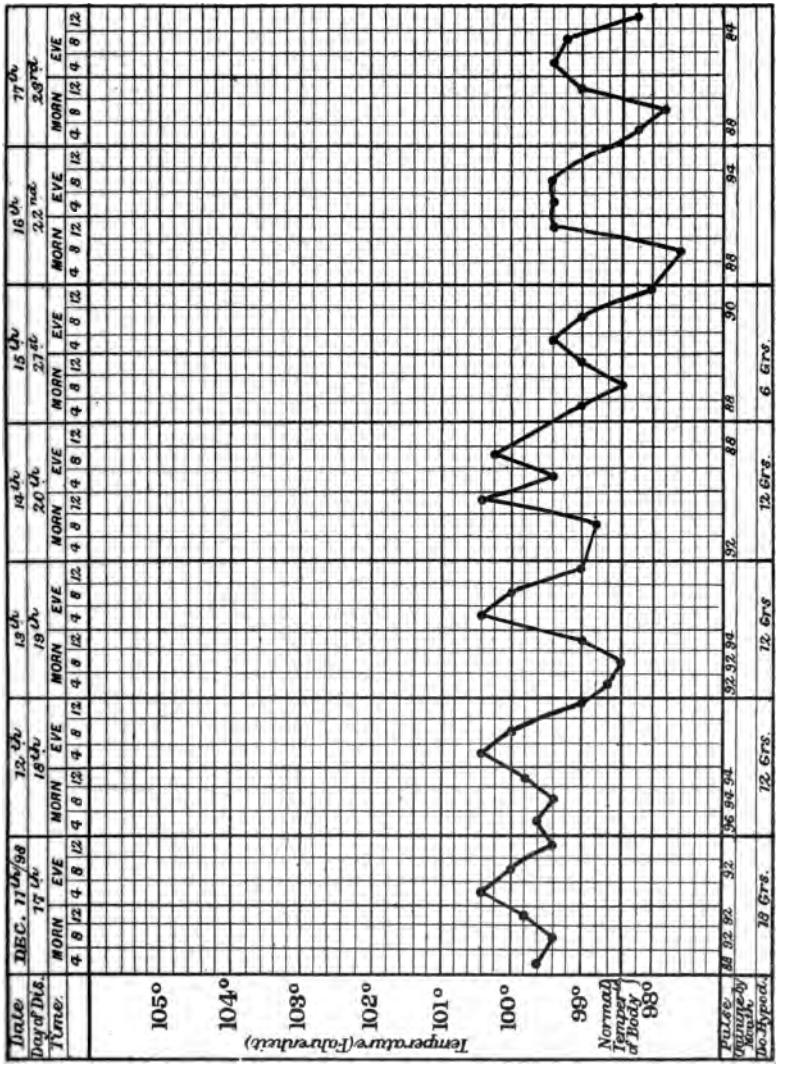
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12 Grs.
6 Grs.

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THE RELATIONS OF BACTERIOLOGY TO EPIDEMIOLOGY.

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MR. PRESIDENT AND GENTLEMEN,

From the remotest ages humanity has been devastated with disease, and imaginative man has ever sought the reason. Ascribed by some to the malevolence of opposing spirits, or the caprice of the "stars in their courses," and other mysterious phenomena of nature, the fancy passed to the machinations of the Evil One, to the craft of witches, or to the chastening hand of an incensed Deity; and even in our own day a relic of this superstition remains in the jural dictum of the "visitation of God." But with the advent of Athanasius Kircher's book, *Pathologia Animata* (1671), and the keen observations of Leeuwenhoek with his microscope (1675), attention was distracted from the occult divinations of astrologers and fetish-hunters to the more proximate, though none the less romantic, study of the agency of invisible organisms. From this time onward the minds of the great tussled with the problems of disease; and an important step was taken when Plenciz, of Vienna, enunciated the specific character of the "*fons et origo mali*," and insisted that each disease was a separate entity in itself, and due to a separate cause. This cause he considered might be transmitted through the air, and falling on susceptible material, there breed and set up its peculiar manifestations. So arose and grew the theory of the "Contagium Virum," till there burst upon the world that glorious controversy upon abiogenesis, or spontaneous generation; which, thanks to the prolonged nature of the discussion, to the acute criticism of its adherents, and to their dogged scepticism, served to place bacteriology on a pinnacle of exactitude never

before attained by a science dealing with biological factors. The researches of this period, of Pasteur, Lister, and Koch, were emphasised by a wave of sanitation which spread over civilised Europe, but most markedly in our own country. Plague, typhus, gaol-fevers, and other pestilences were swept away with the filth of centuries; malaria forsook our shores with the advent of the engineer, and man realised that in these death-dealing diseases he himself had wielded the scourge. Pasteur continued his marvellous work on fermentations, displayed the causes of wine disease, and confirmed and elaborated the observations of Naegeli and Latour on the "spot" disease of silkworms, and later found the cause of the lethargy of silkworms. Thus, from him came the first corroboration of Plenciz's theory of the specific nature of disease, and he was able to show that special organisms were endowed with specific activities: some having the power of setting up disease, others of producing fermentations, and others of causing putrefaction. Lister, at the same time, was studying the causes of suppuration, and laying the foundation-stone of that triumph of all time, the principles of antiseptic surgery. And, while these and other workers were gathering the material upon which to raise the substantial future of the science engendered by the theory of the Contagium Virum, there came from Pollender the description of the first organism found in a disease transmissible to man. In 1849, he described the specific organism of anthrax, and was followed by Davaine; and in 1863 this latter observer established the connection between the organism and the disease: a connection afterwards confirmed and amplified by Koch to its conclusive proof. From that time to the present, under the ægis of Pasteur, Lister, and Koch, disease after disease has been traced to its exciting cause, till now we are conversant with a long list of pathogenic organisms. Some of these have been proved to the full, in others some link in the chain is still wanting, though analogy supplies its place, and renders the issue practically certain whilst others—and these are many—are still shrouded in mystery. But with fuller understanding, more extended powers of observation, and more exact methods of research, every day diminishes the period of their obscuration.

Let us now turn to the relations of Bacteriology and Epidemiology, as they present themselves to us at the present time. And here I would note that by the term "epidemiology", I mean the science which treats of the spread of disease; principally referring to those that are,

or may be, epidemic, but not to the exclusion of those generally endemic. If we take a broad survey of those diseases which are most commonly in occurrence amongst us, it seems that they fall into two great groups, roughly separated by the presence or absence of a marked local lesion.

In the first I would place the acute exanthemata, small-pox, typhus, scarlet fever, measles, rubella and chicken-pox; and to these I would add, as a pendant, whooping-cough and mumps. This group presents certain characteristics. Firstly, they appear to be general or systemic diseases, showing none or little predilection for local action; though I am aware that the sore throat of scarlet fever, the coryza of measles, and the parotitis of mumps may be instanced against this. Still, to me, these are rather concomitants of the disease than evidence of initial and central lesion. Secondly, the diseases in this group conform to one clinical type: an acute invasion with well-marked febrile disturbance, followed for the most part by a characteristic eruption. Thirdly, they are remarkable in being highly infectious, and in occurring almost exclusively in epidemic form. Fourthly, one attack of these diseases confers an immunity of considerable duration, and in many cases of permanency.

The second group comprises those diseases which present a well-marked local lesion, from which, as a centre, the disease as it were radiates. Examples are:—typhoid fever, cholera, diphtheria, erysipelas, suppurative diseases, with pyæmia and septicæmia, tetanus, tubercle, pneumonia, gonorrhœa, glanders, anthrax and rabies. They differ from the first group again in that exanthemata, as a rule, are inconspicuous or absent, and that they present a lower degree of infectiousness, or none at all. They are, for the most part, endemic, though under certain conditions some may become epidemic; and the immunity conferred by an attack is not as a rule so lasting, and in some cases is quite transient.

Between these two groups, as an intermediate class, might be placed plague and influenza—the first with well-defined local lesions extending to the general system; or it may be a general disease, but concentrating in a characteristic manner in certain parts, and capable of conferring considerable immunity; the second, a local disease affecting primarily the buccal mucous membrane, of very low immuning powers, but highly infectious.

Now this division into two groups is, at the present time,

well sustained by bacteriological relation, for, so far, bacteriological methods have entirely failed to discover the *causæ causantes* of any of the diseases in the first group. This is the more surprising, since from their high specificity and their remarkable prevalence, they have attracted the keen attention of numerous observers, and been the subjects of most profound study. True it is that, in many of them, organisms have been described and associated with the disease process; but at the present time we must, I think, consider their specific nature as "not proven." But to uphold for one moment the proposition that for this reason these diseases are not parasitic, would be an anachronism, and not possible to those conversant with the analogies of other diseases. The diseases in this first group are, as already stated, distinguished by a high degree of infectiousness; and this infective matter would seem to be in some measure air-borne, though to what extent such aerial transference can take place is uncertain. In all probability, we shall not be far wrong in considering such powers of diffusion as limited to a very circumscribed area. It is indubitable, however, that the infection may travel by means of an intermediary, such as a person, or clothes, etc., for a considerable distance. But these diseases, as we have seen, are remarkable for their epidemic character: that is to say, the disease is propagated from case to case, and not acquired, so to speak, *de novo*. It follows, therefore, that the infective matter can retain its vitality and powers of setting up disease *outside the body*, but has no power of ectanthropic multiplication.

It may, however, be argued that the infective matter, having lost its virulence, may take on a purely saprophytic existence, till such time as it acquires access of power and can again complete its cycle as a parasite. But to this view there is no evidence in support, and it is distinctly discredited by the fact that nearly all epidemics can be traced to the incursion of infective material from a case freshly arrived amongst the community. Hence the organisms which we assume to be the specific causes of these diseases are obligatory parasites, but capable of resisting external conditions for some little time. It is on this understanding that are based our present methods of coping with these epidemic diseases, namely, by isolation and disinfection, the one to prevent the spread of, the other to destroy, the infective material. For one of these diseases, small-pox, we have a certain prophylactic in efficient vaccination. It is not too much to say that Jenner's

century-old experiments, founded on acute reasoning and carried out in a strictly scientific manner before the bacteriological era, have been the mainspring of sanitary reform and scientific progress; and now by the light of Pasteur's veterinary vaccination, and latter-day researches, have passed from the realm of empiricism to that of secure rationalism.

Of scarlet fever, it was supposed at one time that infection mainly lay in the desquamating cuticle; and although other considerations have lately received more attention, we are still not certain of the cause; and of measles we know that its infectiousness is chiefest in the early stages, whence necessarily the detection is difficult. That the causes of these diseases should so long have eluded the bacteriologist is perhaps surprising, but it may well be, that with illuminated methods, the specific organisms may be found in a category removed from that with which we chiefly associate Pathogenesis.

Turning now to the second group of diseases, it is significant that Bacteriology has something to say about almost all of them; but, before proceeding to enlarge upon this theme, it will be well perhaps to take a brief survey of the present-time attitude of the relations of Bacteria to disease generally. To this end, we cannot err in starting with a recapitulation of Koch's famous postulates. (1) A specific pathogenic organism must be constantly found in the tissues of an animal dead or suffering from the disease in question. (2) It must be found in such diseased tissues only, and not in other diseases or in normal tissues. (3) The organism must be capable of setting up in a healthy animal the same disease process; and from the diseased tissues of this animal the organism must be recovered. (4) The organism should be cultivated outside the body in such manner that it is purified of all other matters, and such culture should be capable of again producing the disease, and the organism be again recovered. (5) This cycle of events must remain constant under similar conditions. (6) The addendum of Martin: that the toxins and toxic products of artificial cultures must be identical with those obtained from the diseased animal.

Now, organisms which conform to these conditions must be considered indisputably to be the causes of the diseases, and, moreover, the specific causes; they can produce one disease and one only, and this invariably. Many specific organisms are familiar to us, such as the bacillus of anthrax, the bacillus of tuberculosis, and that of tetanus.

Without these organisms, there could be no anthrax, no tuberculosis, and no tetanus.

But some organisms are quite wanting in this specific character; thus suppurative lesions may be due to a variety of organisms; and the same organism may be capable of setting up widely different lesions: as witness the diplococcus pneumoniæ in pneumonia and otitis media. It is of the utmost importance, then, to subject an organism associated with a disease to the test of Koch's formulæ, to determine its specific nature.

Again, the idea is gaining ground that two or more organisms can act in conjunction and produce morbid lesions. This is undoubtedly true; but I know of no instance of true symbiosis where the two are absolutely dependent one upon the other; but only of conjunction, where the one lowers resistance, and thereby enables the other to obtain a footing: as happens with the bacillus of tetanus and the staphylococcus aureus. The next important characteristic of the bacterial activity that we have to consider is the bacterial products. It cannot be too emphatically stated that the symptoms of disease are due, not to the specific organisms themselves—unless there be mechanical interference—but to their bacterial products. The consideration of these products is most complex, and this is not the occasion to do more than glance at the questions involved. Bacteria are well known to be capable of setting up fermentative actions, the conversion of starches into sugars, the breaking up of proteids into peptones, and further terms, and so forth. These changes are brought about by bacterial secretions which are called enzymes. Again, bacteria acting on dead proteid matter give rise to more or less poisonous bodies, called ptomaines, and to these at one time were ascribed the symptoms of many infectious diseases. But there was much to negative this idea, and it has now been discarded. Further researches on the bacterial products of pathogenic organisms showed conclusively that other poisons were formed, neither enzymes nor ptomaines, and that these poisons were capable of setting up in a susceptible animal the characteristic symptoms of the disease, apart altogether from the specific organism itself. Attempts to isolate these toxic principles resulted in a shower of names from various chemists, according to the particular phase of chemical fervour at the time, such as albumoses, albuminoid substances, tox-albumins, proteins, nucleo-albumins, and so forth. But it is still doubtful whether these names really

represent the toxic principles, or whether these have really been isolated; indeed, it seems more probable to think with Duclaux, that these poisons are carried down in precipitation, and, as it were, wrapped up in the various albuminous bodies.

At present, then, in speaking of the specific poisons of these pathogenic organisms, it is better to use the simple term, poison, or toxin. But it is proved beyond doubt that the toxins can be separated from the living organisms, and when injected into a susceptible animal can give rise to all the characteristic symptoms of the disease, and lead to a fatal result; and this, with powerful poisons, such as that of tetanus, in incredibly small doses. Here, then, is the explanation of the phenomena of incubation in infective diseases. A person is infected with the living organism: the organism proceeds to grow—slowly or quickly, according to its wont—but the individual feels no ill effects till such time as sufficient toxins have been formed to give rise to constitutional symptoms, and there comes, what we call the invasion, followed by the specific symptoms of the disease.

The action of these poisons is probably different in different diseases, and it is still a moot point whether the bacteria produce the poisonous principles directly, or produce first a ferment which, acting on surrounding proteid matters, gives rise to the specific toxin. Dr. Sydney Martin, in the case of diphtheria, adheres to the latter view, considering that the bacilli secretes an enzyme which induces chemical changes in environing proteids, setting free the true toxins. Buchner and others, on the other hand, consider that the toxins are the direct product of the bacterial cells.

So that the question still hangs in issue as to whether these toxins are true chemical bodies or are of the nature of a ferment. It has already been mentioned that minute doses of powerful toxins are capable of causing a fatal effect in a suitable animal. Moreover, another animal inoculated from this one may also succumb to the intoxication. Further, we know that in such laboratory experiments on animals, there sometimes follows, after introduction of such a minute dose, a latent period—a period as it were of incubation—and then suddenly burst forth acutely the characteristic symptoms. These facts are full of significance, and it would certainly seem in such cases that the toxin possesses a progressive faculty. And if this be so in a laboratory experiment, is it not con-

ceivable that in a highly infectious disease, a minute dose of toxin may pass from the affected individual to the susceptible individual, and there give rise to the disease; that, in fact, the disease may be propagated from one to another by the toxin alone, without the necessary presence of the specific organism itself?

Let us now for a moment turn our attention to the subject of immunity. In laboratory experiments, it is found that where a certain quantity of a culture of a pathogenic organism is required to produce definite morbid lesions and a fatal result in a susceptible animal, a smaller dose will cause only slight local lesion and transient illness, leading to recovery; and a still smaller dose has practically no effect upon the animal. Generally, then, to produce a disease in an animal artificially, the dose must exceed a certain limit. Hence it follows, that animals possess against all diseases certain powers of resistance, or a natural immunity, in many instances extremely slight, but still there. This is noteworthy, because it helps to explain why even susceptible people who may be exposed to infection do not necessarily contract the disease. They may have escaped infection altogether, or, if infected they were able to resist, owing to the smallness of the dose. Infection, then, must be considered in terms of dosage.

There is, however, another side to this, in cases where the tissues, instead of exhibiting a positive power against infection in the shape of immunity, possess the negative tendency of predisposition. In susceptible animals, then, where the natural immunity is very slight, a minute dose may suffice; but with refractory animals, large doses must be administered before they will succumb, and not always then. But by the repetition of sublethal doses, even in susceptible animals, the tissues can acquire powers of resistance, and in this way an acquired immunity is produced. This habituation of the tissues to organisms potentially pathogenic is seen in other ways. Organisms are found existing in a purely saprophytic way in positions where they have frequent opportunities, apparently, of becoming parasitic. The reason that they do not become parasitic appears to be that in organised tissues under normal conditions there exists a natural or acquired defensive power against bacteria and their products. In large part, this would seem to be an acquired property, a faculty of habituation, just as we know that tolerance of the most unfavourable climatic conditions may be acquired on the same principle. This is also illustrated in the fact that the tissues of young

animals are often much more susceptible to the ravages of disease than the tissues of the same animal at a later stage. The young are, as people say, "more tender". This tolerance is well exhibited in our own persons. We know that the superficial layers of the skin and mucous membranes abound with staphylococci, streptococci, and other organisms. These ordinary pyogenic cocci may or may not be lacking in pathogenic properties when inoculated on the usual experimental rodents. But these same cocci have most frequent opportunities of invading the tissues by means of a breach of continuity. How often in shaving does one inflict some slight wound, offering at once an entrance for pyogenic germs. But with what extreme rarity does suppuration result. In nearly all cases, the wound heals at once, and no notice is taken of it. It follows that, under normal conditions, an individual is immune to its own cocci; the tissues have that defensive ability which precludes these organisms from exerting their proclivities. But if the razor be dirty, or foreign matter introduced from without, the case is often different. An organism may be introduced to which the tissues are not habituated, or the defensive powers of these tissues may be weakened by irritation, and there result the phenomena of conflict which we call disease. It is from without, then, that we should anticipate danger, and not from the organisms habitually present with us. Thus in surgery it is of more importance that nothing unclean should come in contact with the wound, than that attempts should be made to sterilise the skin through which that wound is made: in reality a practical impossibility, and apt to lead to a weakened resistance of the tissues most conducive to future mischief.

This defensive attitude of the tissues is further illustrated in relation to the putrefactive bacteria. These organisms, present on the skin and mucous membranes, play a most important part in our economy. They gain, however, no access to the normal tissues, owing to the defensive powers of living protoplasm. But should these powers be weakened or annihilated, straightway the putrefactive organisms seize upon the decaying or dead protoplasm, and exert their wonted functions. Therefore it should not be surprising if, under exceptional circumstances, organisms ordinarily regarded as non-pathogenic develop disease-producing properties. This idea, you may remember, was admirably illustrated by the novelist, Mr. Wells, in his story of the "War with the Martians."

It has already been stated that the characteristic

symptoms of disease may be brought about by exhibition of the toxins, apart from the actual organisms, so also immunity may be acquired by the use of toxins alone; and not only against repeated and larger doses of toxin, but also against the living bacteria. Further, this immunity, by the use of toxins of exalted virulence and in increasing quantities, may be developed to a high degree, but not indefinitely. There is a limit—often a sharp one—and attempts to surpass this fail, causing the immunity to fall, and the animal to weaken and die.

Here we may pause to consider if this is not somewhat analogous to the relapses we meet with in certain diseases, especially typhoid fever. The patient is rapidly improving, has apparently overcome the disease, when suddenly there is a change, and the disease again has the upper hand. Often this is attributed to renewed local irritation, but this hardly meets the bacteriological view of the case; and it seems much more probable that the antitoxin secreting apparatus, working at too high pressure, suddenly collapses, there is a great elimination or metabolism of antitoxin, and the bacillus, before at bay, regains its activity.

The discovery of the immunising powers of bacterial toxins was an immense incitement to work in similar directions, and a mass of information derived from laboratory experiments flooded the bacteriological world. Much of this was imperfect in itself, but, correlated with other facts and other experimental work, proved highly useful. Its value was well seen when Behring was enabled to announce that the blood and serum of an artificially-immunised animal possessed protective properties against the specific organisms of the disease and its toxins; and furthermore, that this serum was also curative, able to arrest the process of disease though fully manifested. The widespread and triumphant effects of the serum treatment, as it is called, which resulted from this discovery, we are all conversant with—they need not detain us here. But as a sequel came another discovery, namely, that such serum exerted a characteristic effect upon the living organisms of the disease; and, moreover, that the action was specific. This property of antitoxic serum was found to be manifested in natural diseases, even at a quite early stage. Hence it was immediately used as a method of diagnosis, and now we know that for one disease, at any rate, typhoid fever, it is a method of the utmost value.

In this, I fear, very imperfect skirmish into the field of

more complex bacteriological problems, I have endeavoured to bring before you such broad principles only as seemed to me to have a more pertinent intimacy with our subject. Rather than exhaust your patience with a discussion on the incomplete experiments which bear on many vexed yet most important questions, I have avoided any reference to them. Indeed, the mass of experimental work performed in the last few years, and daily being added to, on the conjunction of organisms, on their toxic products, and on acquired immunity, baffle the inquirer. Needless to say there oft arises the inevitable query of the flaccid mind, *Cui bono?* No better answer to this can be given than to indicate the splendid success of Behring's serum treatment: a method of prevention and cure which opens before us a vista of incomparable activity; and, again, to point to the value of serum diagnosis.

Now, to revert to Koch's postutales, and to discuss the diseases associated with bacteriological causes by their light. There are several diseases which conform to the requirements there enunciated, namely, anthrax, tetanus, diphtheria, tubercle, glanders, malignant œdema, erysipelas, gonorrhœa, and plague. These diseases, then, may be considered to be of proved bacteriological origin. The organisms are constantly present in the morbid lesions; they are capable of producing analogous lesions in other animals, and can be recovered from these animals; they can be cultivated outside the body, and the pure cultures inoculated into animals produce disease, and from these diseased tissues the organisms can again be recovered. Further, in the case of anthrax, tetanus, and diphtheria, Martin has proved the identity of the toxins of the cultures and the toxins of the diseased tissues. In some other diseases the suspected organisms do not comply with all the requirements; thus neither the organism of relapsing fever, nor that of leprosy, can be grown successfully outside the body. But the blood of a relapsing-fever patient containing the organism has, upon inoculation, produced the disease, whereas inoculation with leprosy material has entirely failed. In influenza, the specific organism has been found and cultivated, but animal experiments are still incomplete, and the same is the case with typhoid fever. In some other diseases there may be several organisms capable of producing the same lesions, and giving rise to similar clinical symptoms. Thus the pyogenic cocci and streptococci may each of them set up a pyæmia or septicæmia, in form clinically indistinguishable. Again, the

same organisms, instead of starting a general disease, may produce only local lesions, and these of very different kinds. Here the lesions may be clinically markedly distinct, but the exciting organism may be the same. In pneumonia, too, the suspected organism is the diplococcus pneumoniæ; but this organism seems often to be associated with other organisms in the production of the disease, and itself is known to be provocative of various other lesions. Indeed, the clinical term "pneumonia" seems to be a convenient cloak which masks several different forms of pulmonary inflammation. The position of cholera I shall advert to later on.

There are many other diseases in which various organisms have been described, but of these, as yet, more evidence is required before they can be seriously accepted.

In still other diseases, apparently of a high specific nature, no organism has been found; for instance, syphilis and rabies. But in regard to the latter, bacteriological methods have stepped in and produced a protective fluid by means of attenuated toxins.

To consider these various diseases *seriatim* in a short paper such as this would be an impossibility. It must suffice, therefore, to make a few comments on some of the more prominent ones.

Diphtheria, as we have seen, answers to the most rigorous tests for a specific organism that bacteriology can devise; and now further proof is found in the specific action of anti-diphtheritic serum. Yet in spite of this, diphtheria is, to some minds, as it were, under a cloud. The reasons for this seem to be mainly twofold—(1) That diphtheria bacilli are found in throats, not clinically recognised as diphtheria. (2) That in cases clinically considered to be diphtheria, diphtheria bacilli have not been found. With regard to the first, all experienced physicians are agreed as to the difficulty at times of diagnosing diphtheria. I myself have often seen, before the bacteriological day, cases planted in various wards of a hospital, because it was not considered advisable to place them at once in a diphtheria ward, and not advisable to put more than one such case in a general ward; and they have been watched, while the physician waited impatiently for albuminuria or other corroborative symptoms. Surely, to an impartial observer, then, the overwhelming bacteriological evidence must quite outweigh clinical diffidence; and it would be considered that if, in an affected throat, diphtheria bacilli are found in numbers, the case *is* diphtheria, although clinically unrecog-

nisable as such. With regard to the second reason, this may bear many interpretations. (1) The case may after all not be diphtheria; (2) the statement that diphtheria bacilli have not been found is of no value, unless we know how often they have been looked for and with what care. It must be remembered that a swab touches only a limited portion of the throat; that by no means all the organisms on the swab are inoculated on to the culture tube; that not all the organisms that grow there can be examined by one specimen or by several; and that not every organism of such specimen can be examined microscopically.

Here, surely, are many opportunities for providing a fallacy, nor are they by any means exhausted. Only the other day, it was my fortune to have sent me a diphtheria swab, carefully taken upon a piece of highly antiseptic wool. It cannot be too emphatically stated, that negative results in diphtheria diagnosis are valueless unless amply confirmed. Nothing can be more foolish, or display greater ignorance, than statements such as "no diphtheria bacilli have been found; *therefore*, the case is not one of diphtheria." It is a stumbling-block to many that diphtheria bacilli have been found in normal throats, and in throats long after convalescence from diphtheria; but these facts rightly viewed are of great usefulness. They serve to show us how the disease may be spread, and how preserved. Moreover, they point to the consistent use of antiseptic mouth washes, not only for the patients but also for those in attendance upon them. But, indeed, the presence of diphtheria bacilli in the healthy throat is of rare occurrence. I have, myself, examined numerous healthy throats, apart from those of individuals in attendance on diphtheria patients, but never yet have I found true diphtheria bacilli—occasionally a few diphtheria-like organisms may be seen in a stained specimen—but that is a different matter altogether. Then diphtheria bacilli have been described in other lesions than diphtheria, fibrinous rhinitis, the rhinitis of scarlet fever, and so on. That these organisms are true diphtheria bacilli has yet to be found; and, in the scarlet fever rhinitis, there is reason to believe that the organism presents some differences. It would be unbecoming, in a general paper of this nature, to embark on a discussion of the various analogues of the Klebs-Löffler bacillus, of its variations and attenuations. Much as has been written about the subject, there is no evidence yet advanced, I think, which militates one whit against the specific relation of the Klebs-Löffler bacillus to diphtheria.

One point that must exercise our minds is the great increase of diphtheria in recent years, and the means by which it has been spread. It is certainly a disease of low infective power, probably transferred only by infected secretions, such as mucus; and the lower animals are not naturally susceptible to the disease, with the exception of cats. The aggregation of town populations and educational activity have had considerable effect, no doubt, but it is questionable if they supply the full answer. Milk is known to be a good vehicle for the conveyance of the infection, and cows have been thought to be responsible for some epidemics. But the presence of bacilli in throats long after convalescence, and occasionally in normal throats, must not be overlooked as indicating a harbourage for the organism, and as constituting possible sources of continued infectiveness. With strict notification and examination of all suspicious throats, much light may be expected to be thrown on the subject; but, although the municipal authorities are displaying some consciousness of this, their methods often leave much to be desired. To give a man £50 a year, and to set him up with test-tubes and an incubator, is not to make an expert of him.

Another possible factor in the spread of diphtheria must not be forgotten, and that is the probable ectauthropic existence of the organism in a manner as yet undiscovered.

There is still deserving of notice one other point, which may in the near future be expected to attract considerable attention: and that is, as to the advisability of issuing regulations in regard to the production of antitoxic sera, with the view of ensuring that all such sera placed on the market shall attain to a certain standard of purity and potency. At the present time, the manufacture of these sera is in the hand of competent institutions or firms. But, with a wider field and increasing demand, commercial enterprise may be expected to start rival establishments, possibly of questionable nature.

The position of the bacillus of *Typhoid* fever as the specific cause of the disease rests assured, since bacteriologists have gained certain methods of distinguishing it from its analogue, the *bacillus coli communis*, the normal inhabitant of the colon. The production of the typical disease in lower animals is still wanting, but cultures of great virulence have been obtained, and animals have been successfully immunised against the disease. Antityphoid sera

have been made and used, but at present with a variable measure of success.

The chief objection raised to the specific nature of the Eberth-Gaffky bacillus is the fact that it is so seldom found in waters suspected of giving rise to typhoid epidemics. That objection, surely, is founded on imperfect consideration. Remembering the long incubation period of typhoid fever, the large volumes of water dealt with, and the difficulties of bacteriological isolation, it would be very surprising if the organism were constantly found. But, at the same time, these waters always show bacteriological and chemical evidence of sewage pollution. There must be very few now who would reject the idea that typhoid fever is a water-borne disease. Hence the discovery of the typhoid bacillus at the height of an epidemic, though interesting to the bacteriologist, is not a matter of supreme importance. It is more essential to advocate such measures as shall safeguard our water supplies from future contamination. It is an easy transition from water-borne typhoid to the infection of food materials, liable to come in contact with water. Milk offers an extremely good medium for the conveyance of typhoid: and in this way, has given rise to epidemics. Oysters and other shell-fish are liable to be contaminated by overflowing sewage as is also water-cress. Before long, it is not improbable that even whitebait and like commodities may come under the ban of suspicion. Here, again, it is more profitable to look to the cause than to lay stress on the multiplicity of effects. Our chief aim should be to see that sewage is directed into proper and harmless channels.

The serum diagnosis of typhoid fever depends on the fact that the specific living organisms lose their power of movement, and become collected into clumps in fluids containing the specific antitoxin. As the blood of the patient contains this antitoxin very shortly after the invasion of the disease, it supplies a ready means for the test. The reaction—like all reactions dealing with biological factors—is not absolute; but it furnishes us with a sign, which seems to be the most constant of all the signs of typhoid fever. How valuable this test may be, is well illustrated in the milk-typhoid epidemic of Clifton, so ably investigated by Dr. Davies.

Influenza is a disease of considerable moment nowadays. The specific organism has been described and cultivated outside the body, but confirmatory animal

experiments are wanting. So far, bacteriological methods have not been largely used to assist in the diagnosis, nor have they assisted in the treatment; and it is doubtful whether much help in these directions can in the future be expected. Certainly, one reason why influenza is at the present time so great a scourge, lies in the fact that it is not treated as an infectious disease. Undoubtedly it is infectious, and probably very highly so. This is a point which requires strongly emphasising. The fact is, the adult population of this country is so unaccustomed to be affected with infectious diseases that the idea is rejected as absurd; and, since the majority of cases are able to get about, any suggestion of isolation is resented. In a way, it is an attitude that one cannot help sympathising with; but, at the same time, it is jeopardising to the health, not only of the affected individual but also of the community at large. Of course, I am not for one moment advocating compulsory isolation —that would be unnecessary and impossible, But I think a great deal would be done if ordinary precautions were taken to prevent the spread of infection, with rigorous attention to disinfection on common-sense lines.

With regard to *Tuberculosis*, the specificity of the organism has successfully withstood the brunt of so much criticism, that it may be looked upon as one of the best-established facts in medicine. There have been many attempts made to treat the disease by means of extracts of bacilli and their products, and by the more recent serum methods, but the results have not been satisfactory. Serum methods which, theoretically, seem the more inviting, have failed because of the difficulty experienced in obtaining powerful toxins, and of producing an adequate degree of immunity.

Tuberculosis must be considered as the product of two factors: (1) predisposition; (2) the activity of the specific bacillus. It is a chronic disease which confers no immunity, which is the same thing as saying that the predisposition remains, though probably it is, at any rate for a time, lessened, by virtue of the struggle which has resulted in victory. The fact that recovery from attacks of tubercular disease does take place, and may be permanent, shows that this factor of predisposition is a plastic one, amenable to treatment. It is true that some authorities discredit the predisposition theory, but the attitude seems untenable, and is opposed to the clinical experience of centuries. We may take it, then, that the predisposition

exists, but by appropriate means it can be diminished and almost nullified. If it were not so, recovery would be practically impossible; and, even if injections of tuberculin and so forth were efficacious against the organism, the patient would speedily succumb to the next chance of infection. Even as it is, it is difficult to see how, in the majority of cases, treatment on the lines of Koch's tuberculin and recent serum methods can be of permanent avail. The organism is so widely distributed, and the chances of infection so numerous, that for the predisposed, with the predisposition a prominent factor, there is but little hope of escape.

The organism grows well outside the body, much better than was originally supposed; and flourishes the more luxuriantly the further it is removed from a parasitic life. It seems certain that it exists outside the body and outside our laboratories, and flourishes as a saprophyte.

True, in this state it is probably much less virulent, and requires passage through several animals before gaining, or regaining, full parasitic powers.

Tuberculous meat and tuberculous milk are fertile sources of infection. Temporary measures, such as proper cooking of meat and the complete or partial sterilisation of milk, are no doubt excellent in their way. But the aim ought to be beyond this. What is wanted is that stringent regulations be enforced, which should ensure the destruction of diseased meat, and prohibit the sale or use of milk from tubercular cows. Such measures would very soon stamp out the disease in cattle. As for patients, isolation is, of course, impossible and undesirable. Much attention has been directed of late to the establishment of country homes for those afflicted. This question has two distinct sides. On the one hand, it is to the advantage of the general community that affected individuals should be removed from it; on the other hand, it is to the disadvantage of the affected that they should be aggregated together. For in such circumstances each individual will practically be placed in a hotbed of infection, and any tendency to recover is likely to be undermined by fresh onslaughts. But, certainly, strict precautions ought to be taken to ensure that all infected material, sputum and so forth, and the rooms inhabited by phthisical patients, be disinfected. Most important it is, however, to combat the predisposition. Much has already been done by sanitary reforms, yet much remains to be done in the way of preventing overcrowd-

ing, of providing efficient ventilation, of furnishing good food, and of encouraging healthy exercise. Open-air *work* for the predisposed, and tuberculosis would be conquered.

The bacteriology of *Cholera* is at the present time in an unsettled state. Ascribed by Koch to one specific organism of comma shape, in the first instance, it has since been found that cholera is due not only to the action of this special organism, but also to that of several other comma-shaped organisms, differing slightly in character from one another, and also from Koch's comma. Now, each of these commas is so far specific, that the anti-choleraic serum of one protects only against the effects of that particular organism, and is useless against the effects of the others. Hence, treatment, whether prophylactic or curative, by means of vaccines or sera, can only be efficacious where the organism producing the disease is identical with that producing the vaccine or serum. The establishment of this identity is a very difficult matter. It is to be hoped, therefore, that further observations on cholera will enable a distinction to be drawn between clinical varieties, and that it may be found that these clinical varieties correspond to distinct organisms. This will greatly facilitate the provision of the appropriate treatment.

With regard to *Plague* I have but little to say. The organism has been found and cultivated. Animal experiments give rise to a disease very similar to that in the human subject. It is known that rats in epidemic times are affected and die of the disease, but whether the disease in them is the forerunner and cause of the human variety, or whether the animals suffer from human infection, is still debateable. The organisms are known also to infect flies, and apparently to cause their death. The question of infected grain or other food-stuffs is an important one, but at present it is merely a question. But there is one matter in connection with plague in this country which is of absorbing interest to the epidemiologist, and that is, what became of the plague after the great epidemic of 1665? It died out almost at once, and the same has been noticed with epidemics in other countries. Its extinction has been attributed to the Great Fire of London; but this could have exerted only a limited

effect, even in London, and not any on the numerous plague-infested districts all over the country.

Quarantine has been cited as the cause, but was in reality not in vogue at the time; and sanitary reforms, however well applied, were not so instant or so thorough as to account for the sudden cessation. Climatic conditions—a cold winter—no doubt helped to stay the disease, though plague epidemics have continued in Russia through the most severe winters. Exhaustion of susceptible material can hardly have been the cause, for thousands of unaffected people returned to the cities on the first sign of a decline. Was the organism annihilated, then, for good and all, or did it take on a saprophytic existence? And if so, is it still with us? A consideration of such questions reminds us forcibly how ignorant we are as to the causes that govern the rise and fall of epidemics.

In speaking of typhoid fever, some allusion was made to the bacteriological analysis of water. This is a matter of considerable moment, in an age of water companies and of the supply of water to an increasing population. Under the old system, water-borne contamination affected only the people deriving their supply from a particular well or wells; now a similar local infection may give rise to a widespread epidemic. It is essential, therefore, that these large volumes of water should be safeguarded from contamination, and should be subjected to efficient analysis at frequent intervals. Much attention has of late years been directed to the methods of bacteriological analysis, and many improvements effected, but the practice is still far from perfect. It cannot be too strongly emphasised that, to obtain reliable information as to the purity of a water, chemical and bacteriological investigation should go hand in hand. The late Sir George Buchanan said that the chemist could tell of impurity and hazard, but not of purity and safety. It is also true that where the bacteriologist can regard only with doubt or suspicion, the chemist can unhesitatingly condemn. An eminent chemist, somewhat sceptical of bacteriology, in chaff, the other day, asked me what would be the value of the bacteriological report, supposing he were to send me for analysis liquid sewage filtered through a Pasteur filter. I said, of course, that if the filtration had been done with care, I should report on the water as germ-free, and as being potable *as far as I knew*; and I asked him, in return what conclusions he would come to if I sent him

distilled water inoculated with typhoid or cholera cultures. These two extremes illustrate very well the necessity of the conjunction of chemical and bacteriological analyses; and I have little hesitation in saying that, of either alone, the chemical is the more accurate and more valuable.

These matters lead us to another subject, which is of great interest to the epidemiologist, and that is, the disposal of sewage. Time does not allow of a consideration of this most important matter; but it seems certain that the trend of thought now is in the right direction, and that the next century will see the utilisation of the agency of that host of organisms, whose function it is to break down organic matter and convert unstable products into stable ones. The rationale of the method is assured, and there is only wanting the experience to devise the best practical application. One important desideratum is, that, besides the thorough and expeditious breaking down of the sewage, the method employed should allow of the recovery of the valuable products of the decomposition, and their return to the land.

Gentlemen, even in this very superficial survey of the relations of bacteriology to epidemiology, I think I have said enough to show that we have considerable ground for congratulation. Bacteriology is only an infant science, but judging from the achievements of the last few years, it is one that may be expected to have before it a long life of immense benefit, not only in the limited field of medicine, but to the general economy. Already it has thrown much light on the nature of disease; it has utilised and elaborated nature's own defences in the demesne of treatment, and it is ever on the track of the sure paths of prevention.

The time may come when the future scientist may see, in part, the fulfilment of the hope expressed by old Omar :—

> "Could thou and I with Fate conspire
> To grasp this sorry scheme of things entire;
> Would we not shatter it to bits—and then
> Remould it nearer to the heart's desire!"

A STUDY

OF

ENTERIC FEVER IN THE NETHERLANDS, DURING THE PERIOD 1875–1894.

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During the last decades the number of deaths from typhoid fever has been, generally speaking, on the decrease. At the same time, the nature and character of the local temporary increase of cases were more closely studied, and an attempt was made to connect the gradual decrease in the death rate with other conditions, such as those depending on the soil, on the movement of the ground water, on the state of the drinking water, on the origin of the milk, on the density of the population and on the presence of hospitals and infirmaries. Great Britain, especially, has done a great deal in this direction. Shirley Murphy, Whitelegge, James Russell, Harvey Littlejohn, and many others, have given extremely instructive accounts of epidemics, and have pointed out with medical certainty the causes influencing the course of the disease. Some very sound and instructive material, with reference to local epidemics of typhoid fever, has, too, appeared from time to time in the Reports of the Medical Officer of the Local Government Board, and also in the *Transactions* of this learned Society, to the members of which I have now the great honour of reading this paper.

Nevertheless, I have chosen deaths from continuous fevers in the Netherlands as the topic of my discourse, on the ground that it must be considered of interest to study at large the increase or decrease of the fever death rate, in order to ascertain if theories on infection, rendered probable by the patient and detailed studies of epidemiologists, are corroborated by statistical methods.

For example, Jenner found that cowpox was a protec-

tion against variola vera. He established this in a number of cases, and the consequence was, the introduction of vaccination into all civilised countries. The manner in which the application of this method had the most widespread results was pointed out by Sir John Simon, in his thorough investigations as to the death-rate of small-pox in Sweden.

Pasteur observed that the chances of recovery in cases of rabies were enormously increased if the victims were treated with a vaccine prepared from the spinal cord of rabid animals. The truth of this statement should be proved by a great diminution in the fatality rate from rabies in those countries where Pasteur's method of treatment has been adopted.

Roux and Behring found that from the toxins present in cultures of the *bacillus diphtheriæ* a remedy could be obtained against diphtheria by injection of the toxine in the blood-vessels of the horse. The great value of this discovery was evinced in an immense decrease of the fatality rate from diphtheria, where patients were treated with this serum.

It is in this way that biological researches are supplemented by statistical data, and that a local and specialised investigation is controlled or verified by that of large populations.

For these reasons, I am of opinion that the statistics of the Netherlands with reference to deaths from fevers will enable us to examine some of the theories which have been advanced as to the causes of the spread of these fevers. But, before discussing the Tables showing you the outcome of my calculations, I must give you some idea of the value of the figures at my disposal. To this end, I shall have to explain in a very few words the working of the Netherlands death register, and in what manner sanitary interests are looked after.

For purposes of administration, the kingdom of the Netherlands is divided into eleven provinces, with provincial councils to supervise the transactions of 1,123 municipal councils. All the 1,123 municipalities, from the largest of half a million inhabitants to the smallest of only 200 inhabitants, are managed practically alike, viz., by a council elected by the male population and a bourgmaster appointed by the Queen. These municipal councils constitute at the same time the sanitary authorities; and they are in their turn supervised, not only by the provincial councils but also by seven special medical government officials. These seven medical officers of health are—with exception of the muni-

cipality of Amsterdam, which has its own medical officer of health—the only official advisers in all hygienic matters. It is also their duty to keep up the medical register, to visit asylums, and to collect the data for the mortality statistics, on the basis of the causes of death. The basis for statistical calculations, viz., the knowledge of the exact number of inhabitants of each municipality, is fairly complete. As everyone whom it concerns is compelled by law to register a birth within three days and a death within five days, and to give notice to the authorities of any change of domicile, the exact number of inhabitants at the end of each year is fairly well known. Moreover, the decennial census which, during the last fifty years has been taken at the end of each decennium, has not brought to light any such serious mistakes as might materially influence the accuracy of the statistical calculations.

The exact number of deaths is similarly well known; and it is the duty of the medical man who attended the deceased during his illness, to acquaint the local authorities to the best of his knowledge with the cause of death. If the deceased was not attended by a medical man during his illness, or in case of sudden death, the authorities request a medical man to hold an inquest; and in supposed violent death, a judge has the power to demand a more extensive investigation.

The causes of death are reported either monthly or, in the case of large communes, weekly, to the medical officer of health, who classifies them. The 34 headings under which the causes of death are entered in our death register do not undoubtedly coincide with our medical views, for the headings being last fixed in 1875, were arranged not only on scientific principles, but also—and justly so—from a practical point of view. However much an anatomical system, or any other scientific system, may be laid down as a foundation, yet medical men will fill in only that which they wish to convey; and thus a number of headings which, for instance, would only be filled up in such places as hospitals are simply ignored by the general practitioner.

It is, therefore, necessary to have a certain knowledge of the opinion of men of the profession: and this is difficult, as, owing to the rapid strides medical science has made within recent years, a practitioner of 25 years' standing holds quite different and often opposite views on momentous questions of the day to those of a recently-qualified man. For—as far as my paper is concerned—there is to be

noted that there is still a heading for typhus and typhoid fevers, for continuous fever, and another for intermittent fevers.

The inquiry is further favoured by the fact that, since 1875, "the rubrics of the causes of death" have remained unchanged.

Again, be it mentioned, that since the Act for the prevention of the spread of infectious diseases was brought into operation, at the end of the year 1872, every practitioner is compelled to give notice of any case of infectious disease that may come under his observation.

Both typhus and typhoid fevers are included in Art. 1 of this Act; and one might reasonably expect that, since 1872, the number of registered cases would throw full light on the knowledge of the spread of these diseases: the more so as almost every community in my country appoints a parish doctor whose duty consists in looking after the patients of the poorer classes, and even in visiting them at their homes. Experience, however, teaches us that medical men will register only such cases as infectious which they really think to be so, and consequently, as a certain number of them are of opinion that enteric fever ought never to have been included in the Act of 1872, they fail to notify cases of this disease. Moreover, any house harbouring a sufferer of any infectious disease has pasted on it in a prominent position a notice paper, upon which in large letters is printed the name of the disease. Such a house is naturally avoided, and any business carried on on the same premises must suffer materially. So it happens that many a medical man considers rather the financial interests of his patient than the law, especially where typhoid fever is concerned.

Consequently, the figures giving the number of cases of typhoid fever, as published in the yearly medical reports, are of no value.

But are, then, the return of deaths from typhoid and typhus fever of any value at all? Is it not conceivable that the physician who fails to give notice of the case of typhoid fever will also omit to fill in the true cause of death, in order to avoid inquiries from the medical officer of health: who, as the blank is not only filled in with the cause of death, but also contains the domicile, age, sex, vocation, etc., can easily inquire very closely into the matter? Some explanation is due to you on this point, especially if I tell you that ever since the introduction of the Bill on Infectious Diseases, there was noticed a sudden

and apparently important decline in the percentage death rate from typhoid and typhus fevers.

As I mentioned before, the rubrics of the death statistics were altered in 1875, but the heading "Typhus and typhoid fevers," appeared in the registers of 1866-1874 as well as those published after 1875.

Since 1875, however, there was also inserted a heading, "*febris continua*;" and if we now calculate the death cases from typhus and typhoid fevers during 1865-1898, per 10,000 inhabitants, we note also an enormous decrease for the years 1873 and 1874. Table I shows this clearly. Take now the number of deaths from "*febris continua*" since 1875, also calculated per 10,000 inhabitants, add to them the death cases from typhus and typhoid fevers, and we find that the decline continuing regularly after 1875 is almost to a same degree as that before the introduction of the Bill. (See Table I, p. 174.)

The regular decrease in the death-rate from typhus and typhoid fevers before the passing of this Act on the one hand, and from all continuous fevers after the passing of the Act on the other, has induced me to combine, in all my further calculations, the death cases from the two groups. This naturally gives rise to errors; but these would certainly have been more numerous had typhus and typhoid fevers only been taken as a group by itself.

There exists, however, another and greater difficulty, viz., the separation of deaths of typhus from those of typhoid. Many physicians mean by typhoid: slight cases of abdominal typhus, and by typhus the *status typhosus* with unconsciousness and delirium. The observations of of the temperature, which in all doubtful cases is of the greatest diagnostic value, are not—or at any rate, were not—generally taken into account in the practice amongst the poor, nor, in some cases, amongst the rich. Owing to the difficulty in the differential diagnosis in some cases, there were, and perhaps there are still, physicians who think there are transition forms, as appears from the reports of the medical officers of health.

If now we estimate as near as possible the death-rate from typhus during the period 1875-1895, and define this disease by taking the cases of such an infectious nature as to attack the medical and nursing staffs, as *typhus exanthematicus*, then we find that deaths from this disease are about 8 per cent. of the total deaths from typhus and typhoid fevers taken together. This would be all very

well had these deaths from typhus been more or less equalised over the four five-yearly periods; but this evidently is not the case, as the number of deaths, especially in the northern provinces, was in the first period far above

TABLE I.

Deaths from Typhus, Typhoid Fever, and Continuous Fever, per 10,000 living.

—	Typhus and Typhoid Fever.	Continuous Fever.	Both Combined.	—
1866	10.7	—	—	
1867	9.1	—	—	
1868	10.0	—	—	
1869	9.0	—	—	
1870	9.0	—	—	
1871	8.0	—	—	
1872	7.9	...	...	Infectious Diseases Act, 4th Dec., 1872.
1873	**5.0**	—	—	
1874	**3.3**	—	—	
1875	3.4	3.7	**7.1**	New Headings of the Death Register.
1876	3.4	3.2	**6.6**	
1877	2.7	3.0	**5.7**	
1878	2 9	2.9	**5.8**	
1879	2.0	2.6	**4.6**	
1880	2.4	2.8	**5.2**	
1881	2.2	2.7	**4.9**	
1882	2.0	2.1	**4.1**	
1883	1.8	2.1	**3.9**	
1884	1.8	2.1	**3.9**	
1885	1.3	1.8	**3.1**	
1886	1.5	1.7	**3.2**	
1887	1.3	1.4	**2.7**	
1888	1.3	1.2	**2.5**	
1889	1.4	1.2	**2.6**	
1890	1.3	1.1	**2.4**	
1891	1.4	1.1	**2.5**	
1892	1.5	0.9	**2.4**	
1893	1.2	0.7	**1.9**	
1894	1.0	0.6	**1.6**	
1895	1.1	0.5	**1.6**	
1896	1.0	0.5	**1.5**	
1897	0.9	0.4	**1.3**	
1898	1.0	0.3	**1.3**	

that in the other periods. On this account, a certain though unavoidable error has crept into my calculations. However, the number of deaths from all forms of continuous fever, to which I will confine myself, is mainly due to typhoid fever.

I shall now endeavour to point out to you different causes which have influenced this diminished death-rate; and the Tables which I have compiled as the result of my investigations show the mean annual death-rate in the four five-yearly periods, viz., 1875-79 80-84, 85-89, 90-94, and to which I will refer as periods I, II, III, and IV.

We might feel inclined to attribute the diminished fever death-rate to such general improved conditions as would lessen the death-rate as a whole.

This is, however, not the case, as Table II will show. The decline in the general death-rate, which from 23.3 in the first period came down to 20.0 per 1,000 living souls in the fourth period shows an improvement of *more than* 14 per cent.

Deaths due to tubercular disease of the lungs and hæmoptysis diminished from 226 to 192 per 100,000 living souls: hence a diminution of 16 per cent.

Deaths from continuous fevers, typhoid included, diminishing from 60 per 100,000 to 22, show a decline of 63 per cent.; and lastly,

Deaths from intermittent fevers, descended from 17 per 100,000 to 2, a diminution of 87 per cent.

TABLE II.

Enteric Fever in the Netherlands.

Period.	Death-rate per 1,000 living.	Deaths from Phthisis per 100,000.	Deaths from all Continnous Fevers per 100,000.	Deaths from Intermittent Fevers per 100,000.
I.	23.3	226	60	17
II.	22.2	202	44	10
III.	20.6	191	28	6
IV.	20.0	192	22	2

The conclusions drawn from these figures are therefore to the effect that although general causes, which also seem to have influenced tubercle of the lungs, undoubtedly have brought about a diminution in the number of deaths, yet other circumstances must have acted upon the death-rate from continuous fevers, whilst malaria at the present time can hardly be considered as an interesting cause of death at all in the Netherlands.

Or, differently expressed: it seems that typhoid fever as a preventable disease *par excellence*, has yielded more favourably to the improved conditions of the times than other diseases.

Influence of Density of Population.

Dividing the kingdom into large and small communes, we will endeavour to trace the influence exercised by the growth of the towns upon the death figure in general, and upon that of continuous fevers more in particular. For this purpose, we have to take into consideration several points.

The opinion that density of population is an unfavourable health factor is still maintained, and on the whole justly so. There are, however, a goodly number of exceptions to this rule. For example, Dr. Newsholme showed that the death-rate in the densely-populated labourers' model dwellings, the Peabody Buildings in London, was less than the average total death-rate met with in London.

We must, however, not lose sight of the fact that the larger municipalities, generally the towns, with their more detailed administration and with their better financial position, are infinitely better capable of carrying out hygienic improvements than the small communes in the country. Again, towns have more medical men available; medical aid is more easily obtainable; admittance to hospitals, infirmaries, etc., is carried out with greater facility, than in the small country places; where, if hospitals do exist at all, they can hardly be regarded as on a line with their sister institutions in the towns and larger communes.

Which of the influences are now the weightier? The unfavourable conditions, as offering a greater scope for infection by means of the density of population and the uncertain origin of food, especially milk; or the favourable influences, with their superior medical aid and their greater expenditure for hygienic purposes?

In the first period, about 1,103,811 people = 28.3 per cent. of the total population, were living in communes with more than 15,000 inhabitants; whilst in the last period the figure was 1,649,260, or 35.4 per cent.: that is a relative and an absolute increase in the population of the towns.

In the towns the general death-rate diminished from nearly 26.2 per 1,000 to 20.6 per 1,000, or a diminution of fully 21 per cent.; and in the small communes from 22.1 per 1,000 to 19.6, or a diminution of only 11.3 per cent.

Although always still a little higher than in the country, the general death-rate is diminishing to a greater degree in the towns, and no doubt the large towns will soon have the best of it altogether.

On the other hand, the death-rate from continuous fever has, during the four five-yearly periods, always been much lower in the towns than in the country; moreover, the diminution in the towns is of greater import. The number of deaths from continuous fever in the towns fell from 50.8 per 100,000 living souls to 14.6, or a gain of 71.3 per cent.; whilst in the country the figures diminished from 61 to 25.4 per 100,000, or an improvement of nearly 60 per cent.

We are therefore quite justified in stating that in the Netherlands the inhabitants of towns run less risk of dying from typhoid fever than those living in the country, and that this risk is growing less still. With very few unimportant exceptions, the truth of this statement is evident in all the provinces of the kingdom.

Table III shows the figures for the communes of more or less than 15,000 inhabitants, divided in different groups according to their population. I hope to have pointed out clearly enough that, judging from the total death-rate during the four periods, a decided improvement in the general health condition may be deduced, especially for the towns, and that the fever death-rate is lower in the towns than in the country. The favourable factors of town life appear thus to outweigh the unfavourable conditions due to density of population. (See Table III, p. 178.)

Influence of Drinking-water.

The death-rate from typhoid fever in the Netherlands being lower in the towns than in the country, the question arises, whether this favourable result might not in the main be due to the better state of the drinking water in the towns? The water question in the Netherlands is, indeed, a very difficult problem. Taking into consideration the fact that the most flourishing parts of the kingdom lie below the level of the sea; that the soil, from a geological point of view still very young, is permeated by salt water, and on account of its low-levelled position is incapable of replacing the salt water by rain water: it is easy to understand that, up to fifty years ago, the inhabitants of some of the most flourishing towns and villages collected rain-water in underground reservoirs for drinking purposes. In dry summers, however, the inhabitants of these rain-water

TABLE III.

A.—*General Death-rate per* 1,000 *in Groups of Communes according to the Population.*

Period.	Amsterdam [308-429,000].	Rotterdam and the Hague [246-387,000].	Utrecht [67-88,000]	Towns with Populations of 25,001-50,000 [266-486,000].	Communes with Populations of 15,001-25,000 [223-259,000].	*The Foregoing Combined.*	Communes with Populations of 5,001-150,000 [762-1,134,000].	Communes with Populations under 5,001 [2,036-1,870,000].	*The Last Combined.*
I. ...	25.2	26.4	26.1	25.4	28.0	**26.2**	24.8	21.1	**22.1**
II. ..	25.5	21.4	25.6	25.4	21.4	**24.0**	20.2	21.3	**20.9**
III. ...	22.4	22.4	23.6	23.2	23.2	**22.5**	19.9	19.8	**19.8**
IV. ...	21.2	20.6	21.4	21.0	20.2	**20.6**	19.3	19.7	**19.6**

B.—*Deaths from all Continuous Fevers per* 100,000 *living.*

I. ...	60.4	37.6	42.7	61.6	41.6	**50.8**	62.9	63.3	**63.1**
II. ...	49.4	23.6	44.0	39.4	46.6	**39.6**	40.9	47.4	**45.2**
III. ...	20.1	20.4	21.4	21.4	25.2	**21.4**	27.6	33.3	**31.3**
IV. ...	19.7	13.1	19.9	17.6	10.4	**14.6**	20.3	28.5	**25.4**

communes are very soon obliged to drink water from canals and ditches, and so expose themselves to all the dangers attached to the consumption of such water.

In the higher situated parts of the land, good drinking water can, as a rule, be obtained from the ground; and, whereas communes on a river used in olden days to provide themselves with unlimited quantities of untrustworthy river water, at the present time a good many of them supply the inhabitants with filtered river water by means of a network of pipes. The first ground-water system was laid in 1853 for the towns of Amsterdam and Haarlem by an English company, which introduced water from the sand-dunes on the coast of the North Sea; in 1856 the naval harbour of Helder followed suit; and Rotterdam, the second town in the kingdom, only decided twenty years afterwards, viz, in 1874 on a river-water system. After that, the call for an ample and pure drinking water supply became more general. During the period I, seven towns, with a population of 534,000 inhabitants, or 13.6 per cent. of the total population, had or came in the possession of a ground-water system from the sand-hills or the heath. During the fourth period, twenty-two towns and sixteen smaller communes, with an aggregate population of 1,329,000 inhabitants, or 28.5 per cent. of the total population, enjoyed the same benefit.

Rotterdam, with 140,000 inhabitants, or 3.5 per cent. of the total population of the kingdom, was during the first period the only town with a river-water system. Seven towns and five small communes—435,000 inhabitants, or 9 per cent. of the total population—had a similar river-water-system in the fourth period.

There is always considerable difficulty in ascertaining the exact number of inhabitants who do really avail themselves of this water system. The water companies make, for instance, contracts with the landlords for the supply of water to their property; in other cases, again, the leaseholders, or even the tenants of single flats, enter into a contract with the water companies. Some houses are not connected at all; and thus it happens that the tenants, especially those residing in the poorer quarters, have to fetch their drinking water from the standpipe in the street, or from a house where water is sold.

The condition of affairs in this respect, in the several towns and villages under my observation, cannot accurately be determined nor described in a few words. I know, for instance, a village—Assendelft—having only one standpipe

belonging to a water company, which, in order to supply a neighbouring town, had to lay the pipes on Assendelft territory. The inhabitants of the village generally drink rain-water; but if during a dry summer their supply of rain-water becomes exhausted, barrels of water from the standpipe are sold to the villagers for a trifle.

All the same I have included this village among the municipalities connected with a water system; for the reason that the very existence of a good water supply evidently obviates, in case of need, the necessity to make use of the polluted and impure water of rivers and canals.

The situation of these two groups of communes is given on the map, while the figures are to be found in Table IV.

The most extensive group: communes with ground-water systems shows a death figure which, from 25.2 per 1,000 living souls in the first period, fell to 20.4 in the fourth, or an improvement of 19 per cent. The communes with a river-water system had a higher death figure, which from 27.3 per 1,000 in the first period irregularly diminished to 20.9 in the fourth period, showing a gain of 23.5 per cent.

Continuous fevers demanded, on an average, in communes with ground-water systems, 60 victims per 100,000 inhabitants annually in period I: this number was, in period IV, only 19, hence a diminution of more than 68 per cent.; whereas, for communes with a river-water system, the death rate from continuous fevers in three out of the four periods was considerably lower than that in ground-water communes, and also fell from 32 to nearly 15 per 100,000; hence a diminution of nearly 54 per cent.

TABLE IV.

Period.	Communes with a Water-supply from the Sandhills.		Communes with a River-Water supply.	
	General Death-rate per 1,000.	Deaths from Continuous Fevers per 100 000.	General Death-rate per 1,000.	Deaths from Continuous Fevers per 100,000.
I.	25.2	60	27.3	32
II.	24.9	44	22.4	30
III.	22.2	21	23 2	28
IV.	20.4	19	20.9	15

The influence of drinking water, as deduced from the

above figures, on the death rate from continuous fevers, does not seem to be very preponderant; anyhow, not so clear as the influence exercised by the size of the communes.

Furthermore, the result is not in accordance with accepted views. Although ground water is much less exposed to pollution than river water, yet we find that the death rate from typhoid or continuous fever in communes with river-water systems is lower than in those possessing—from a sanitary point of view—a much more favourable water condition.

A *provisional* explanation of the fact that communes, on introducing a ground-water system, experienced a very large improvement in the death-rate — now almost approaching the always still lower fever death rate in communes with a river-water system—is readily at hand. In communes on rivers, a sufficient quantity of fresh water is always to be obtained, independent of water systems. A number of communes with a ground-water system were, however, frequently suffering from a want of water. Now, want of water means want of cleanliness; and it is not at all unlikely, that *and* the better quality, *and* a more adequate quantity of the drinking water supplied, as well as the more liberal use of it, have greatly assisted in bringing down the death-rate from fevers. The striking fact remains, however, that in communes with a ground-water system, the death-rate from general causes is lower, whilst the mortality due from continuous fevers is higher, than in the river-water communes. This phenomenon impresses us even more forcibly if we consider that Asiatic cholera, a disease propagated specially by drinking water, has in the Netherlands played the greatest havoc along the course of the rivers.

Dr. van der Meulen found that in Amsterdam deaths from typhus and typhoid fevers had diminished from 191 per 100,000 during 1854-59, to 21 per 100,000 during 1880-89; whilst for the same periods the number of contracts for water supply had increased from 4 to 70 per 1,000 inhabitants. Accurate data, however, as to exact quantities of water supplied per head, the more general demand for it in the municipality, the number of persons who during each period remained true to the old sources of water provision, are difficult, if at all obtainable, for all municipalities provided with a drinking-water system.

The great significance of a sound drinking-water system has, however, been discussed so frequently in connection

with typhoid fever, and also appears so very probable to my own mind, that I could not refrain from bringing to your notice the above-mentioned calculations, which, I must confess, do not by any means clearly show the influence of a healthy drinking water.

Influence of the Soil.

The influence of the soil on the spread of typhoid fever has, since the historical researches of Buhl and Pettenkofer, alternately been accepted and rejected in numerous writings. The strange coincidence at Munich, of an increase in the number of deaths from typhoid fever with a concurrent fall of the level of the ground water, has induced many other towns, with more or less success, to look for similar phenomena. We have not a clear notion, however, in what manner the spread of typhoid fever among man is influenced by certain conditions of the soil. Air currents are not capable of bringing bacteria which have devoloped in a polluted soil from below to the surface.

Excavations of the soil certainly cause outbreaks of malarial fever in those districts where the disease is prevalent; but to attribute a great spread of typhoid fever solely to the mere digging up of the soil is not a generally accepted medical view. Even the multiplication of pathogenic bacteria in the natural soil seems to occur very rarely, and then only under exceptional conditions. Generally they disappear after a few weeks, and on the strength of this bacteriological fact it is once more difficult to believe in the direct influence of the soil on our health, as far as typhoid is concerned.

We are rather inclined to think that the influence of the soil acts indirectly: that by circumstances depending on the condition of the soil, but not on the soil itself, our health is influenced. A population nourished by a good productive soil enjoys a greater prosperity than one supported by a poor and barren soil. This poor and barren soil will of necessity be scantily populated, and hence the risk for infection will be less; on the other hand, however, medical assistance will be much more difficult to be obtained than in the flourishing districts. In this way it can be explained that towns favourably situated from a commercial and industrial point of view, although built on an unhealthy soil, are nevertheless capable of showing a lower death

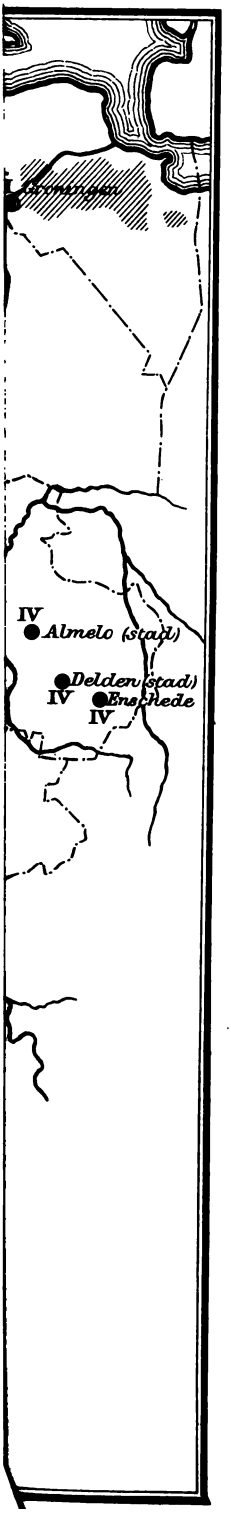
IV
Almelo (stad)
Delden (stad)
IV
Enschede
IV

figure than those places built on a dry soil, but devoid of business enterprise.

The Netherlands may now be divided into two parts, depending on the character of the soil, viz., one part situated below and another situated above high-water level. The former would be inundated in case of a flood, the latter would not. The low-lying part of the country is, in a sense, artificial, inasmuch as it was dyked in in the course of centuries, and is, even at the present time, continually being relieved of its superfluous water by means of wind- or steam-mills. This superfluous water is partly due to the rain, and, in the case of recently dyked-in portions, partly to water, which forces its way underneath the impermeable clay dykes.

The accompanying map shows (shaded) that portion of the Netherlands which lies below the level of the sea. The map shows, at the same time, that our large cities, such as Amsterdam, with a population of half a million inhabitants, Rotterdam, the Hague, and Utrecht, must be included either entirely or partially among the low-lying portion of the kingdom. From this it appears that the low-lying portion is the most flourishing part of the kingdom. Large cities arise only there, where much money can be earned. The greater value of the low-lying districts may also be ascertained from other data. Wherever live-stock can be reared, landed property will generally be of greater value than in districts suitable for agricultural products only. The breeding of cattle requires a humid soil, and a uniform humidity is secured by the uniform level of the water in the polders. The province of Groningen, mainly containing land above the level of the sea, is the only province which can show high rents for landed property equal to those of the low-lying provinces, especially the two provinces of Holland: at an average, about £6 per H. A. =£3 per acre.

Taking further into consideration the fact that every town increases the well-being of the outlying suburbs, by raising the prices of agricultural products, and thus enabling the suburban population to profit by it greatly, I think that I have proved clearly that the low-lying districts, which after all contain the greatest number of large towns, are undoubtedly the most flourishing.

This flourishing condition must of necessity affect most favorably the death-rate in general, and perhaps also that of continuous fevers. On the other hand, there are several disadvantages in connection with the low-lying districts

due to their position. Heavy structures have to be built on piles driven into the earth. The peaty upper soil is too marshy and too mobile for laying sound foundations. The result is, that the houses are built as lightly as possible, the walls thin, and the entire building feeble. Very old buildings are a rarity here: the mobility of the ground causes comparatively new houses, even those the foundations of which are laid down in a deeper and more concrete subsoil, to bulge forward.

Again, the low lands have no running water. The two main branches of the Rhine flow, united with the Meuse, between high dykes—protecting the country behind them—past Rotterdam towards the sea. The other so-called rivers of the low lands, as the Amstel, the Zaan, the old Rhine, and the Vecht are in reality only navigable fresh waters separated from the sea by locks, and in which the level of the water falls and rises according as water flows through these locks into the sea, or from the polders into these so-called rivers. In Amsterdam, Zuider-sea water is run into the "grachts" which traverse the town, during the night, in order to refresh them by means of artificial currents.

The low lands are thus artificially drained, and nature aids man very little in this case for, with the exception of most of the towns and some of the villages, the low land is dependent for its domestic use on small quantities of rain water, preserved in reservoirs.

Owing to the want of running water in the low districts, sewage, dirty water, and factory refuse are got rid of in a very unsatisfactory manner. It is very difficult indeed to remedy this evil. This vital sanitary problem is still far from a satisfactory solution everywhere, the metropolis even not excepted. In Amsterdam the greater part of the sewage runs through drain-pipes into the canals; another smaller portion is removed by the pneumatic system of Liernur; another by carts from a part of the houses, twice a day: early in the morning, and late in the evening. In small towns, and in the villages, the sewage is thrown into ditches and canals. Agriculture cannot make a proper use of it in cattle-breeding districts. The disinfective action of the soil can therefore not show itself, and the more or less intense pollution of stagnant—or at any rate of only temporary running—water is solely the outcome of the situation and character of low lands. This is a perpetual source of anxiety to the authorities, and of discord among the inhabitants.

In following the result of these favourable and unfavourable conditions, we have to compare the general death-rate, and that of continuous fevers of communes lying below the level of the sea, with that of the dry districts. A group of communes situated in part on low and in part on high land has to be considered separately. To be exact, the large communes, viz., those with more than 15,000 inhabitants, have to be excluded. They lie, as we have mentioned above, for the greater part in the low lands, and show as such a lower death-rate for continuous fevers.

In the period I, 1,844,318 people were living in small communes, the territories of which were situated above the level of the sea; 534,663 in the small communes of the low lands; and over 419,000 in mixed districts. These figures have increased in the course of the twenty years —the period of our investigation—but not uniformly so, for the three groups. The percentage of the total Netherland population, living in small communes, fell during these twenty years from 71.7 per cent. to 64.6 per cent. Relatively, therefore, the towns have a greater increase than the country. The small communes in the high lands contained 47.3 per cent. of the total population during the first period, and 42.2 per cent. during the last period, the country population of the low lands and mixed districts combined on the other hand diminished from 24.4 per cent. to 22.4 per cent. for the same periods, and shows therefore a relatively smaller loss than the high lands. It is possible that the economic conditions of the dry districts are the less favourable, and thus may have led to a greater immigration into the large towns.

Table V gives the death-figure and mortality from continuous fever in the four periods for small communes lying above or below, and also of those lying partially above or below, the level of the sea. By this we see that the death-figure during the twenty years for the small communes in the high lands has not greatly improved.

The improvement for the other two groups, however, is clear. For the communes lying in part high and in part low the improvement is nearly 19 per cent., and for the communes of the low lands more than 23 per cent. (See Table V, p. 186.)

This important fall of the death-rate, especially in the case of the inhabitants of the low lands, who showed in the first two periods a clearly higher rate than those living on

the high lands, points strongly to the generally improved conditions of life and to a steady progress.

The mean annual mortality from continuous fevers has during the four periods been invariably greater in the low lands: next in order are the communes, situated in part on high and in part on low land; but the high lands showed the lowest death-rate of all. It further strikes one that, during these twenty years, the mortality from typhoid fever has diminished, with an almost equal percentage for all the groups of small communes, viz., with fully 61.8 per cent. for the low districts, 61.2 per cent. for the mixed communes, and with 58.5 per cent. for the high lands. The main conclusion to be drawn from these figures is, that in those parts of the low lands not offering exceptionally

TABLE V.

Period.	Small Communes lying *below* the Sea-level.		Small Communes lying *partly below* the Sea-level.		Small Communes lying *above* the Sea-level.	
	General Death-rate per 1,000.	Deaths from Continuous Fevers per 100,000.	General Death-rate per 1,000.	Deaths from Continuous Fevers per 100,000.	General Death-rate per 1,000.	Deaths from Continuous Fevers per 100 000.
I.	25.4	88.4	21.8	68.6	21.2	54.4
II	24.6	66.3	19.8	50.2	20.1	38.1
III.	20.8	42.4	18.8	34.0	19.6	27.2
IV.	19.5	33.8	17.6	26.6	20.0	22.6

favourable conditions, as, for instance, the rise or growth of large cities, the risk of dying from typhoid fever is greater than in the high lands. The germ of infection seems to enter the intestinal tracts of the inhabitants with greater facility, owing probably to the markedly defective sewerage system.

The uniform improvement in the mortality from this disease throughout the total number of small communes in the Netherlands, whether living on natural soil or on artificial "polder land," makes one think that one or more general causes must have tended to bring about this state of affairs. The sewerage system in general has not been much improved upon, and certainly not at all in our small communes; a small number of villages only are connected with a drinking-water system; and hospitals, although

greatly changed for the better in the towns, are scarcely to be met with in the small communes.

To assume that a large proportion of the population in the course of time has become immune to typhoid infection seems to be very improbable indeed, if we take into account the relatively insignificant cause of death with respect to the general mortality. On the contrary, it may be that the virulency of typhoid fever has abated.

It seems to me, however, much more likely that the earlier and better recognition of the disease has led to a better and sounder treatment. We have learnt that slight cases may give rise to severe attacks in others. Again, the study of bacteriology has spread among the medical profession a clearer conception of the modes of infection. In the towns, where the scope of action of the profession is greatest, and the opportunities offered for the application of their science most abundant, there the gain during the twenty years has been 72 per cent. In the country, however, where for obvious reasons the practice of medicine is liable to greater inconveniences, and subject to numerous unavoidable difficulties, there, independent of the high or low situation, the improvement for the same period amounted to 60 per cent.*

Typhoid fever may justly be considered a preventible disease, if proper measures of a general sanitary nature are taken. The prevention of the disease rests in part on the authorities. The diminution of the mortality, however, is in great part due to the spread of hygienic ideas among the laity, and the early recognition or suspection of the disease by the profession.

The physician endowed with the knowledge of a modern university forms an important—if not the most important —factor in the prevention of infection; especially in diseases, such as typhoid fever, which, if sufficiently early recognised, offer on the whole a not-unfavourable prognosis.

* This diminution of the death-rate from typhoid in the Netherlands is in accordance with the same phenomena in Germany and in England.

OBITUARY.

Sir William Jenner, Bart., G.C.B., M.D., F.R.S.

Sir William Jenner died on December 11, 1898, at his residence, Greenwood, Bishops Waltham, Hants, at the age of 84. He had retired from public life about ten years previously, and had for some time been suffering from the growing infirmities of age. But the announcement of his death recalled to mind his unique personality and the great services he had rendered to medicine, of which he was, by common consent, the recognised leader in this country. Nor can lapse of time efface his claims to remembrance: he has left too many behind him who have been indebted to his wise counsel for health, to his luminous teaching for knowledge, to his upright example for conduct. This is not the place to sketch, even in outline, the history of his life; that has been amply done elsewhere. Suffice it to say that, beginning in obscurity, he won for himself the highest position open to a British physician, by means of his indomitable energy and devotion to his life-work, as much as by his unswerving integrity.

The early years of Jenner's professional life were exceptionally arduous, and were spent in general practice; but he was not content to remain in this humble position, and having obtained the M.D. degree of London University in 1844, he set himself to work out problems of etiology and pathology, which were then engaging the minds of physicians in America and France as well as in this country. The great question was as to the nature of "fever;" and already, by Stewart in Glasgow, Gerhard in Philadelphia, and Louis in Paris, more or less apprehension of the truth as to the specific distinctness of typhoid fever had been attained. But the doctrine was not as yet held generally, and it required the minute and painstaking analysis to which Jenner subjected it to settle definitely the problem. For upwards of two years he devoted himself almost entirely to this labour, making and recording clinical and pathological observations upon every case admitted into the London Fever Hospital, of which his friend Dr. Sankey

was then resident medical officer. The material which he thus collected with scrupulous care was first published to the world in 1849, in a series of papers contributed to the *Edinburgh Journal of the Medical Sciences*, and subsequnetly issued in book form. The republication of this and his other writings on the same subject in 1893 (Rivingtons') enables the student of to-day to realise what a vast amount of labour and fine powers of investigation were required to produce such work. This publication, as well as the paper read at a meeting of the Royal Medical and Chirurgical Society on December 11, 1849, and the detailed clinical notes which appeared in the *Medical Times* in the following year, may be said to have settled once and for all the absolute non-identity in origin, symptoms, and morbid characters of typhoid and typhus fevers; and yet at the meeting referred to, several leading physicians who shared in the debate expressed doubts as to the validity of the arguments. The following passage from the preface to the volume issued in 1893 is worth quoting in this regard. Sir William Jenner there says that "all the facts detailed and analysed were observed and recorded at the bedside and in the deadhouse by myself. While collecting some of these facts, in 1847, I caught typhus fever, and three or four years later typhoid fever. I mention this because it was said at the time, 'Before typhus and typhoid fevers can be said to be absolutely different diseases, someone must be found who has suffered from both;' and I was the first, so far as I know, who at that time could be proved to have suffered from both. Dr. E. A. Parkes attended me in both illnesses, and had no doubt about the diagnosis in each case."

Sir William Jenner was admitted to the membership of the Royal College of Physicians in 1848, and in 1852 was elected to the fellowship and was chosen Gulstonian Lecturer. He selected the subject of "Acute Specific Diseases" for his lectures, which are also reproduced in the above-named volume. It was not until 1863 that he became a member of the Epidemiological Society, and in 1866-68 he occupied the Presidential chair. His inaugural address will be found in vol. iii (O. S.) of our Transactions. Its main theme was the prevention of diseases; and in it he pointed out how much there was to be done in respect to the determination of the causes and conditions under which zymotic diseases prevail; and he emphasised the duty of the Society in respect to their investigation, "to collect evidence, to disseminate knowledge, to spur on those in power

to frame laws adequate for the occasion; to note every local outbreak of epidemic disease; and, either as individual members of the Society or by committees, to investigate the causes of such outbreaks, and then to spread the knowledge thus gained." It is gratifying to know that the honour bestowed upon him in the session 1897-98 by the Society in electing him an honorary member was much appreciated by Sir William Jenner, whose services in many departments of medicine had during his long life been often similarly recognised.

It only remains to add that in 1848 he was appointed Professor of Pathology at University College, and also assistant physician to its hospital: becoming in 1854 full physician, and some years later, professor of medicine; that he was the first physician appointed (in 1852) to the Hospital for Sick Children, Great Ormond Street; and that in 1853 he was elected to the staff of the London Fever Hospital. In 1861, on the death of Dr. Baly, and on the advice of Sir James Clark, he was appointed Physician-extraordinary to the Queen, and in 1862 was made Physician-in-Ordinary, an office he held until 1893. He was created a baronet in 1868, K.C.B. in 1872, and G.C.B. in 1889. Her Majesty has put on record the esteem and friendship which she had for Sir William Jenner; whilst amongst his professional brethren the regard in which he was held was made apparent when, with general approval, he so worthily occupied the high office of President of the Royal College of Physicians from 1881 to 1888.

S. C.

The Ordinary Meetings of the Society are held at

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THE CHAIR IS TAKEN AT HALF-PAST EIGHT O'CLOCK P.M. PRECISELY.

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